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AIRBLOCK IN THE NEWBORN PERIOD*

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INTRODUCTION

In recent years there has been a great deal of interest in the efficiency of the lung as an air container. It has become apparent that air may escape from the respiratory passageways in a wide variety of conditions in all age groups.[1] The resulting clinical syndrome has been termed airblock.[2] I have been particularly interested in this problem as it applies to the newborn period.

On large newborn services it is a well-appreciated observation that in a high proportion of the deaths the cause of the fatal outcome may not be discovered at autopsy. All too frequently the death is attributed to small areas of atelectasis, although it is well known that normally the lungs may require several days or more completely to expand; to intracranial hemorrhage of insignificant size; to "immaturity"; to an enlarged thymus; or to other causes of questionable importance. The purpose of the investigation of my colleagues and myself[3, 4] has been to determine if airblock might be a factor in some of these deaths: either alone in severe cases, or in less severe instances in conjunction with other factors which of themselves might not have led to a fatal outcome. It should be stated here that small amounts of aberrant air may cause little harm and produce no symptoms.

There are two good reasons for believing that aberrant air is frequently unappreciated at necropsy. The first is that it may be easily overlooked. Pneumothorax may not be detected unless particularly sought for. Although emphysema in the anterior mediastinum is readily detected, in the posterior mediastinum it is easily missed. Pulmonary interstitial emphysema may pass unrecognized or be thought an artefact. If the lung is fixed by immersion of small blocks of tissue, the air may be forced out by shrinkage caused by the fixative. The second reason is that it has been demonstrated clinically that aberrant air is not uncommon: one variety, pneumothorax, occurs in approximately one per cent of newborn infants.[5] Regardless of the cause of death, pneumothorax might be expected to be present in one per cent of newborn deaths. Moreover, if pneumothorax is ever deleterious in itself, it might be expected to be present in more than one per cent. Such an incidence of pneumothorax suggests that other varieties of aberrant air may not be unusual. These two reasons seem to warrant the inclusion of efforts to detect aberrant air as a part of the routine postmortem examination of newborn infants.

ETIOLOGY

Both experimental[2] the clinical [4, 6] evidence indicates that the escape of air from the respiratory passageways is in the lung itself. Pulmonary interstitial emphysema

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is the primary pathology. Experimental evidence[2] indicates that the site of escape is through the bases of alveoli which lie upon the smaller blood vessels. This escape is likely to take place (a) when alveoli are overinflated without corresponding increase in the circumference of the underlying vessels, or (b) when the circumference of the vessels is decreased without reduction in the size of the alveoli. Increased alveolar pressure in either instance increases the likelihood of escape of air. The caliber of the blood vessels may be reduced in several types of congenital heart disease or by forced expiration leading to reduced return of blood to the right heart and correspondingly to the pulmonary circulation.

Clinical conditions which may lead to the production of pulmonary interstitial emphysema are not uncommon in the early newborn period. These include atelectasis of a degree to be pathological; any type of congenital heart disease which decreases the volume of pulmonary blood flow; the irregular respiration sometimes caused by intracranial hemorrhage; excessively vigorous methods of resuscitation; and aspiration of meconium, mucus, or excessive amounts of amniotic fluid. Normally, the expansion of airless lungs, necessary to establish respiration, requires a much greater negative pressure than does normal respiration, probably because of cohesion of the moist surfaces of the air passages in the collapsed state.[7] Moreover, extensive changes in both the cells lining the alveoli and the connective tissue of the lung occur with expansion of the lungs. When all of these factors are considered it becomes apparent that the ability of the respiratory passageways to act as an airtight container receives a severe test in the newborn period.

PATHOLOGY

I have examined a small series of newborn infants coming to necropsy for various reasons and found pulmonary interstitial emphysema to be quite common.[8] It is difficult to draw any conclusion, however, because so many of the infants have had artificial respiration which may lead to a false high incidence. Nevertheless, I believe pulmonary interstitial emphysema to be quite frequent in amounts insufficient to cause significant harm. Less frequently it is present in larger amounts and may cause hemorrhage in the lung; splint the lung in a state of inflation, thus limiting the respiratory excursion; and by its perivascular location lead to a partial block in the pulmonary circulation. Some of these changes are seen in figures 1 and 2.

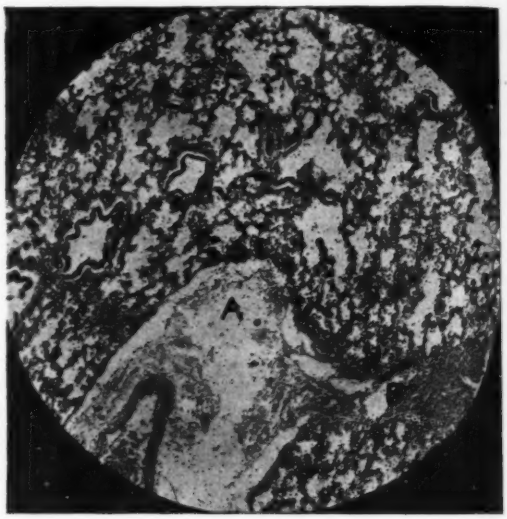

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Fig. 1: Photomicrograph (approximately x116) of a section from the lung of a newborn infant dying of airblock. At A is a large perivascular collection of air. Note the hemorrhage which extends beyond B along a connective tissue plane.

There is excellent clinical evidence that in newborn infants interstitial emphysema may rupture the visceral pleura directly to produce pneumothorax: of six infants with airblock coming to autopsy, pulmonary interstitial emphysema and pneumothorax were present in all, yet mediastinal emphysema was present in only one.[4] In three of these six patients the pulmonary interstitial emphysema was particularly marked near the hilus of one or both lungs, suggesting that this may be the site of rupture of the visceral pleura. When pneumothorax is produced it frequently develops a pressure higher than atmospheric, probably because

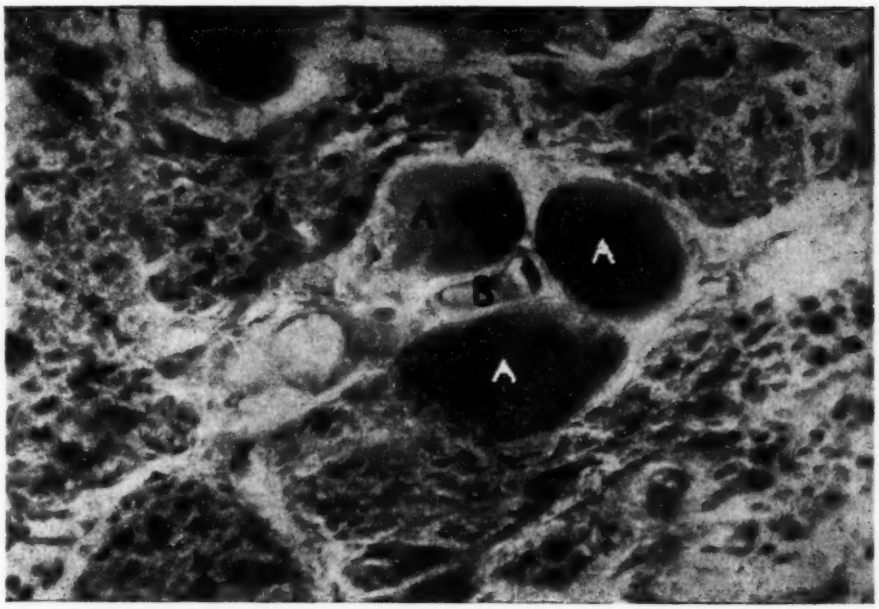

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Fig. 2: Very low power magnification (approximately x15) of the cut surface of the fixed lung of a newborn infant dying of airblock. At A are large collections of air surrounding a blood vessel, B. Note the generalized ectasia. Some of these collections of air are in normal passageways, but many are in the interstitial tissue.

of strong expiratory effort "pumping" air into the pleural space.

Interstitial air in the lung may not rupture the visceral pleura but may travel along the great vessels to the mediastinum to produce mediastinal emphysema.[6] If a pneumothorax has already been produced by rupture of the visceral pleura and the site of this rupture is near the lung root (as above suggested), there is no reason why mediastinal emphysema may not also be produced: as the pressure in the pleural cavity rose there would be an increased likelihood of air continuing into the mediastinum. From the mediastinum the air may rupture the mediastinal pleura to produce pneumothorax;[9] theoretically, the pneumothorax could be either on the same or the opposite side, or even on both sides. The deleterious effect of mediastinal emphysema in newborn infants has been widely described. However, it is not always easy to determine what portion of the harm is due to the mediastinal emphysema and what to the pulmonary interstitial emphysema. Nevertheless, it can be said that in the newborn infant with mediastinal emphysema, and no doubt pulmonary interstitial emphysema, aspiration of the former will relieve alarming symptoms.[9]In animals, where mediastinal emphysema without pulmonary interstitial emphysema can be produced experimentally, the harmful effects of the former are probably due to pressure of the air on the great vessels, particularly the large veins.[10]

From the mediastinum the air can travel upward into the cervical subcutaneous tissues or downward along the great vessels into the retroperitoneal tissues to produce pneumoperitoneum. Extension of the air to extrathoracic locations is not a serious complication and may actually be accompanied by clinical improvement, the pressure

inside the chest having been diminished.[1]

There are two aspects of the subject which need further investigation. The first is the effect of pulmonary interstitial emphysema upon the heart. When extensive, because of its perivascular location, pressure on the vessels, and consequent block to the pulmonary circulation, it must lead to cor pulmonale. The second is the probability of air embolism.

SYMPTOMATOLOGY

There is no report, to my knowledge, of an autopsy on a newborn infant in which the sole finding which could have caused the death was pulmonary interstitial emphysema. For this reason an authoritative correlation of pulmonary interstitial emphysema with a definite clinical picture cannot be made. I suspect, however, that many of the symptoms and signs mentioned below, which have been seen in infants having pulmonary interstitial emphysema with mediastinal emphysema and infants having pulmonary interstitial emphysema with pneumothorax, can be caused by pulmonary interstitial emphysema alone. These symptoms are, of course, seen in only the more severe cases; as already mentioned, small amounts of aberrant air probably cause little harm and produce no symptoms.

The outstanding symptoms of mediastinal emphysema with pulmonary interstitial emphysema are dyspnea and cyanosis. In the more severe instances the chest becomes expanded and in spite of marked effort the infant is able to no more than partly expire. Consequently the respiratory excursion is greatly reduced, although the respiratory rate may be considerably increased. The neck veins may become visibly distended. The area of mediastinal and cardiac dullness may be diminished. The heart sounds may be faint and distant; the crackling crunching sound synchronous with the heart beat that has been often described in adults is less frequently heard in newborn infants. Although adults may complain of pain, this symptom is difficult to evaluate in the newborn period. If palpable cervical subcutaneous emphysema appears, the diagnosis is greatly reinforced.

The symptoms of pneumothorax with pulmonary interstitial emphysema are similar in many respects to those of mediastinal emphysema with pulmonary interstitial emphysema. Dyspnea and cyanosis are common to both. Similarly, in the more severe instances the infant may have an expanded chest and expiration be difficult and limited. The neck veins may be distended. The cardiac findings, however, are different. Instead of distant heart sounds and the crunching sound, the heart is likely to be shifted either to the right or to the left. Over the pneumothorax the characteristic changes in percussion and auscultation may be obtained. Cervical subcutaneous emphysema does not occur.

If pulmonary interstitial emphysema, mediastinal emphysema, and pneumothorax—all three—are present, all of the symptoms and signs mentioned in the two preceding paragraphs may be present.

X-RAY FINDINGS

Although a roentgenographic diagnosis of pulmonary emphysema may offer little problem, the differentiation of compensatory from obstructive emphysema may be more difficult. Inspiratory and expiratory films are required;[11] in infants these are difficult to obtain accurately. Moreover, once a diagnosis of obstructive emphysema is made, it may be impossible to decide whether the major portion of the air is on the inside or outside of the normal passageways. If escape of air from the normal pathways is as frequent as my experience indicates, both compensatory and obstructive emphysema, more particularly the latter, are frequently complicated by interstitial emphysema. For this reason any marked emphysema of recent acute onset may lead to suspicion of interstitial emphysema.

A roentgenographic diagnosis of mediastinal emphysema is not always easy. An anteroposterior or postero-anterior film of the chest may show a curvilinear shadow of air along either margin of the mediastinum or along the border of the heart. A lateral view may show a large collection of air in the anterior mediastinum. However,

neither of these views is absolutely reliable; extensive mediastinal emphysema may be present yet these views fail to show it. If a pneumothorax is also present the value of the lateral view is further reduced: air in the pleural cavity, particularly if under tension, can produce on the lateral film a shadow identical with that produced by air in the anterior mediastinum.[4] Moreover, it is likely that severe emphysema in the anterior portion of the lung may also cause an apparent substernal collection of air. This would be particularly true if the film were slightly overexposed.

The roentgenographic characteristics of pneumothorax are well known. It should be emphasized, however, that there may be no relation between the size of a pneumothorax and its pressure. A comparatively small pneumothorax may be under very high tension as evidenced by increased distance betwen the ribs, depression of the diaphragm and shift of the mediastinum. If the underlying lung contains sufficient interstitial air, it splints the lung in an expanded position and, being trapped there, prevents more than moderate collapse. Similarly, if the opposite lung is involved, it may be so expanded as to prevent shift of the mediastinum, although the pneumothorax of the opposite side is under very high tension as evidenced by widely spaced ribs and depressed diaphragm.

Some of the x-ray changes mentioned above are illustrated in figure 3.

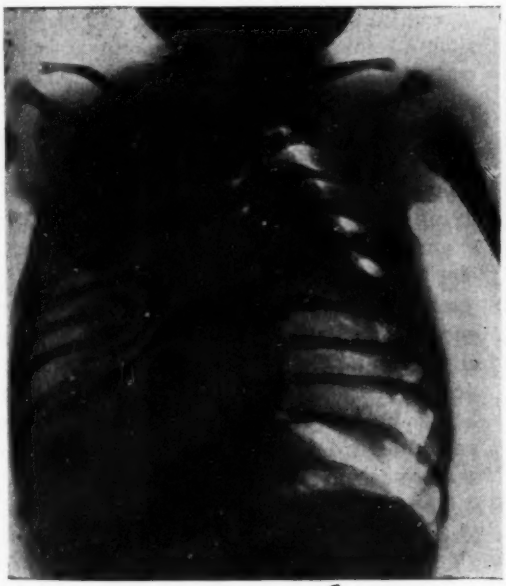

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Fig. 3: X-rays of the chest of a newborn infant with airblock who died a few hours later. Reproduced from the *Journal of Pediatrics* (4) by permission of the editors. A: Anteroposterior view. There is atelectasis of the right upper lobe. The left pneumothorax is evidently under great tension. Note the widely spaced ribs, shift of the mediastinum, and depression of the left diaphragm. Yet the left lung is only partially collapsed. Further collapse is prevented by pulmonary interstitial emphysema, air trapped in the tissue of the lung. B: Lateral view. The apparent substernal collection of air is not due to mediastinal emphysema; there was no air in the mediastinum at autopsy. The shadow is due to herniation of the anterior mediastinum by the tenison left pneumothorax.

TREATMENT

A review of the known etiologic factors suggests that measures designed to avoid the occurrence of airblock must be largely a matter of the best obstetric attention possible, together with careful resuscitation of those infants needing such measures. If extensive atelectasis suggests the advisa-

bility of bronchoscopic aspiration, this procedure is feasible in the newborn period.[12]

It is likely that in most instances the amount of aberrant air is small, causes few or no symptoms, and needs no treatment. In many more the amount of aberrant air is so small as to produce only mild symptoms; in such cases the only indicated treatment is the administration of oxygen and gentle care of the infant.

In more severe cases some effort to remove portions of the aberrant air may be advisable. If a large or tension pneumothorax occurs, aspiration may be indicated; this may have to be repeated several times. Gumbiner and Cutler[9] have reported the alleviation of alarming symptoms following aspiration of the anterior mediastinum in a newborn infant with mediastinal emphysema. An effective therapeutic approach to pulmonary interstitial emphysema has not been devised.

REFERENCES

1. Macklin, M. T., and Macklin, C. C.: Malignant interstitial emphysema of the lung and mediastinum as an important occult complication in many respiratory diseases and other conditions: an interpretation of the clinical literature in the light of laboratory experiment, Medicine, 23:281, 1944.

2. Macklin, C. C.: As quoted by No. 1 above.

3. Salmon, G. W., and Forbes, G. B.: Pneumothorax in the newborn infant, J. Pediat., 23:50, 1943.

4. Salmon, G. W., Forbes, G. B., and Davenport, H.: Airblock in the newborn infant, J. Pediat., 30:260, 1947.

5. Davis, C. H., and Stevens, G. W.: Value of routine radiographic examinations of the newborn, based on a study of 702 consecutive babies, Am. J. Obst. & Gynec., 20:73, 1930.

6. Fisher, J. H.: Spontaneous pulmonic interstitial and mediastnal emphysema in an infant, Canadian M. A. J., 44:27, 1941.

7. Wilson, J. L., and Farber, S.: Pathogenesis of atelectasis of the newborn, Am. J. Dis. Child., 46:590, 1933.

8. Salmon, G. W.: Unpublished date.

9. Gumbiner, B., and Cutler, M. M.: Spontaneous pneumomediastinum in the newborn, J. A. M. A., 117:2050, 1941.

10. Jehn, W., and Nissen, R.: As quoted by No. 9 above.

11. Caffey, J.: Pediatric x-ray diagnosis, Chicago, 1945, The Year Book Publishers, p. 244.

12. Woodward, F. D., and Waddell, W. W., Jr.: Bronchoscopy in the newborn infant, J. Pediat., 23:79, 1943.

POSTMORTEM OBSERVATIONS IN TWENTY-TWO PREMATURE INFANTS*

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This paper is not intended to be a statistical survey of the causes of death of premature infants dying during the neonatal period. Adequate reviews of this subject, based on autopsy findings, have been presented in the papers by Potter and Adair,[1] Potter,[2] Cruickshank,[3] D'Esopo and Marchetti[4] and Macgregor.[5]

Although pathologic findings, presumably indicative of the cause of death can be demonstrated in 70 to 88 per cent of viable premature infants dying in the neonatal period, many clinicians and pathologists still feel that autopsies on premature infants are of little value, as they consider prematurity alone to be an adequate cause of death. The responsibility for this misconception rests both with the clinician and the pathologist. The clinician has been lax in obtaining permissions for post mortem examinations on premature infants dying in the neonatal period, and the examinations performed by the pathologist on such infants are usually inadequate. The present paper is presented to encourage better cooperation between the clinician and the pathologist in dealing with premature infants. This cooperation is badly needed, for there is probably no period of life during which clinical diagnoses are so unreliable.

The material for the present paper consists of the consecutive autopsy records of 22 premature infants dying during the neonatal period on the Tulane Service of the Charity Hospital of Louisiana at New Orleans. The deaths occurred between October, 1945 and May, 1946. All the infants were born alive, weighed more than 499

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grams and less than 2500 grams, and died between five minutes and 25 days after birth. Twenty were colored and two were white. Complete postmortem examinations including examination of the contents of the cranial cavity were carried out on all infants. Bacteriologic studies were inadequate since most of the bodies were unclaimed and the autopsy, by law, could not be done until 48 hours after death. All the autopsies were performed by the author.

Among the 22 premature infants five were classed as previable (weight 500-999 grams). An adequate cause of death was demonstrated in four of these. There were 17 classed as viable premature infants (weight 1000 to 2499 grams). An adequate cause of death was demonstrated in all of these infants (Table 1).

In the entire group of 22 infants 16 died in the first 24 hours after birth, two died between 24 and 48 hours and four died between 14 and 25 days (Table 1). All of the previable premature infants died within 48 hours and only one lived over 24 hours.

The causes of death in the previable premature infants were bronchopneumonia, two, and intracranial hemorrhage, two. No cause of death could be demonstrated in one infant weighing 904 grams and living one hour and 36 minutes. The causes of death in the viable premature infants were congenital syphilis, five, bronchopneumonia, four, asphyxia, four, thrombosis of the venous sinuses of the dura mater, two, and intracranial hemorrhage, two.

In the entire group of 22 infants bronchopneumonia was the immediate cause of death in six, congenital syphilis in five, asphyxia in four, intracranial hemorrhage in four, dural sinus thrombosis in two and the cause of death could not be determined in one. Infection, including congenital syphilis, was held accountable for 11 of the 22 deaths (Table 1).

In the 16 infants dying during the first 24 hours there were four cases each of bronchopneumonia, congenital syphilis, and asphyxia, three of intracranial hemorrhage and one in which the cause of death could not be determined. Only two infants died between 24 and 48 hours, one of an intraventricular hemorrhage and one of congenital syphilis. There were two infants with bronchopneumonia and two with dural sinus thrombosis who died between 14 and 25 days (Table 1).

These figures are too small to be of any statistical significance but they do show that even in premature infants important pathologic changes can be demonstrated by careful postmortem examinations. The detailed findings should be of interest to obstetricians and pediatricians.

Bronchopneumonia was present in six cases as the immediate cause of death and occurred in infants dying from 17 minutes to 25 days after birth. It was present as an incidental factor in four others, three dying of congenital syphilis and one of lateral sinus thrombosis. The intensity of the process varied considerably, being more severe in the infants who lived over 48 hours. In these cases it probably represented a postnatal infection. Three of the four infants dying during the first 24 hours of bronchopneumonia showed microscopic evidence of aspiration of amniotic sac contents. In two infants the inflammatory reaction was slight and asphyxia may well have been more important than pneumonia as the immediate cause of death. Macgregor[6] believes that some cases of pneumonia with prenatal aspiration may be caused by a non-bacterial irritant in the aspirated material. In 541 consecutive autopsies on infants up to 28 days old, she found inflammatory changes in the lungs in 37 per cent of the live-born infants and in 11.8 per cent of the dead-born. Premature infants appeared to be especially susceptible to respiratory tract infections. Johnson and Meyer[7] believe that a clinical diagnosis of congenital pneumonia is practically impossible, and that nearly all infants dying of pneumonia within three days after birth do so as a result of antenatal infection.

Congenital syphilis accounted for five deaths in the present series. All of these occurred in viable premature infants dying between one hour and 30 minutes and 34

hours after birth. Congenital syphilis may manifest itself only by osseous changes (Schmidt,[8] Schneider[9]) but in the present series, although bony changes were constant, other evidences of congenital syphilis were also present. These included hepatomegaly and splenomegaly, syphilitic pancreatitis and in one infant extensive fibrosis of the hypophysis. A similar change in the hypophysis has been described by Browne[10] who found syphilis to be the most important form of antenatal infection. Potter,[1] however, found congenital syphilis in only two of 954 autopsies on infants dying during the intrauterine and neonatal periods. Cruickshank[11] has pointed out that syphilis in the mother is not an important cause of abortion but is an important cause of premature delivery. The appalling incidence of congenital syphilis in the present series, in spite of the small number of cases, demonstrates the continued importance of syphilis as a public health problem, at least among the Negro race in the New Orleans area.

Asphyxia accounted for four of the deaths. All were viable premature infants dying in the first 24 hours. There is still disagreement as to the occurrence of intrauterine respirations in the normal human fetus. Small amounts of amniotic sac contents are often found in the lungs but it is generally agreed that a large amount is indicative of fetal asphyxia. Amniotic sac contents in the lungs may be identified by the presence of cornified epithelial cells and also by a pink staining coagulum applied to respiratory surfaces—the so-called asphyxial membrane (Johnson and Meyer[7] "hyaline membrane"; Farber and Sweet[12] "vernix membrane" and Baar[13]). Cornified epithelial cells derived from the skin of the fetus may be entrapped in this membrane or may lie free in the bronchioles and alveoli. In the lungs of the four infants in the present series whose deaths were attributed to asphyxia there were keratinized epithelial cells in the air spaces and a pink amorphous membrane lining many of the bronchioles and alveoli. Wilson and Farber[14] believe that actual bronchial obstruction due to aspiration of the contents of the amniotic sac is less frequent in premature than in full term infants, as the production of vernix and cornification of the skin take place largely in the last months of pregnancy.

In addition to the four infants in whom death was attributed directly to asphyxia, eight others showed evidence of aspiration of amniotic sac contents varying from slight to moderate. In these cases asphyxia may have been a contributory cause of death. Two infants showed a few small foci of subependymal encephalomalacia in the white matter neighboring the lateral ventricles. Such lesions may be the result of asphyxia or of birth injury, but in themselves did not appear to be important factors in causing death. These focal subependymal necroses have been carefully described by Schwarz,[15] and Benda[16] has reported similar lesions in the brains of mentally deficient children.

Intracranial hemorrhage was responsible for four of the deaths. One infant showed an extensive subarachnoid hemorrhage and scattered petechiae in the substance of the brain; two showed subarachnoid, intraventricular and small intracerebral hemorrhages and one showed an intraventricular hemorrhage and petechiae in the brain. Two of the four cases occurred in previable premature infants. The duration of life varied from five minutes to 31 hours. In no instance was there an associated tear in the falx or tentorium. There is a difference of opinion as to whether intracranial hemorrhages without gross dural tears are the result of birth trauma or of asphyxia, and Holland[17] has pointed out the difficulty in distinguishing between traumatic and asphyxial intracranial hemorrhage.

Thrombosis of the dural sinuses was responsible for two deaths. Both occurred in viable premature infants living 14 and 18 days. No local inflammatory basis for the thrombi could be demonstrated. In one infant both lateral sinuses were thrombosed and in this case there was an associated bronchopneumonia. In the other infant there were thrombi in the superior sagittal

and lateral sinuses and in the great vein of Galen. This infant also showed subarachnoid, intracerebral and intraventricular hemorrhages. Bailey and Hass[18, 19] and Byers and Hass[20] have emphasized the importance of acute nutritional disturbances in the pathogenesis of dural sinus thrombosis in early life.

Table 1

Status of Prematurity

	Previable Duration of Life			Viable Duration of Life			
Cause of Death	1-24 hrs.	25-48 hrs.	14-25 days	1-24 hrs.	25-48 hrs.	14-25 days	Total No.
Bronchopneumonia	2			2		2	6
Intracranial hemorrhage	1	1		2			4
Congenital syphilis				4	1		5
Asphyxia				4			4
Sinus thrombosis						2	2
Undetermined	1						1
Total by length of life status	4	1	0	12	1	4	
Total by status		5			17		22

DISCUSSION

It cannot be denied that prematurity was an important contributing factor in the death of many of these infants. Prematurity, for example, favors the occurrence of such lesions as intraventricular hemorrhage.[5] It is also true that the full term infant may survive trauma and infections which might kill the premature infant. Thus obstruction of scattered bronchioles and alveoli may have little effect in the full term infant, owing to the relatively large pulmonary reserve. In the premature infant the total functional pulmonary parenchyma may be barely sufficient to maintain life and minor obstruction in the respiratory tract can be fatal.

The purpose of this paper is not to belittle the importance of prematurity as a factor in neonatal deaths but rather to show that if death occurs serious lesions can be demonstrated as the immediate cause in most instances. It is encouraging that many of the deaths were not the inevitable consequence of prematurity but were theoretically preventable.

SUMMARY

An adequate cause of death was demonstrated at autopsy in 21 of 22 premature infants dying during the neonatal period. Bronchopneumonia and congenital syphilis together accounted for 11 of the deaths. Other causes of death were asphyxia, intracranial hemorrhage and dural sinus thrombosis. These findings indicate that prematurity uncomplicated by other disorders is seldom the sole cause of death.

REFERENCES

1. Potter, E. L., and Adair, F. L.: Clinical-pathological study of the infant and fetal mortality for a ten-year period at the Chicago Lying-in Hospital, Am. J. Obst. & Gynec., 45:1054, 1943.

2. Potter, E. L.: The lessons to be learned from a study of infant deaths, J. A. M. A., 124:336, 1944.

3. Cruickshank, J. N.: Child life investigations: The causes of neonatal death, London. H. M. Stationery Office, 1930. (Med. Res. Council Special Report Ser., No. 145.)

4. D'Esopo, D. A., and Marchetti, A. A.: The causes of fetal and neonatal mortality, Am. J. Obst. & Gynec., 44:1, 1942.

5. Macgregor, A. R.: The pathology of still-birth and neonatal death; A review of 1053 cases, Brit. M. Bull., 4:174, 1946.

6. Macgregor, A. R.: Pneumonia in the new-born, Arch. Dis. Childhood, 14:323, 1939.

7. Johnson, W. C., and Meyer, J. R.: A study of pneumonia in the still-born and new-born, Am. J. Obst. & Gynec., 9:151, 1925.

8. Schmidt, M. B.: Syphilitische Knochenveränderungen der Neugeborenen, Ergebn. d. allg. Path. u. path. Anat., 7:262, 1900-01.

9. Schneider, P.: Die angeborene Frühsyphilis im Knochensystem, die osteochondritis und Periostitis syphilitica congenita, und ihren Beziehungen zur Spirochätenverbreitung, Virchows Arch. f. path. Anat., 234:378, 1921.

10. Browne, F. J.: Discussion on stillbirths and neonatal deaths. IV. Neo-natal death, Brit. M. J., 2:590, 1922.

11. Cruickshank, J. N.: Discussion on stillbirths and neonatal deaths. V. Syphilis as a cause of antenatal death, Brit. M. J., 2:593, 1922.

12. Farber, S., and Sweet, L. K.: Amniotic sac contents in the lungs of infants, Am. J. Dis. Child., 42:1372, 1931.

13. Baar, H. S.: The postmortem examination of the newborn infant, Brit. M. Bull., 4:178, 1946.

14. Wilson, J. L., and Farber, S.: Pathogenesis of atelecasis of the newborn, Am. J. Dis. Child., 46:590, 1933.

15. Schwartz, P.: Die traumatischen Schädigungen des Zentralnervensystems durch die Geburt. Anatomische Untersuchungen, Ergebn. d. inn. Med. u. Kinderh., 31:165 1927.

16. Benda, C. E.: The late effects of cerebral birth injuries, Medicine, 24:71, 1945.

17. Holland, E.: Birth injury in relation to labor, Am. J. Obst. & Gynec., 33:1, 1937.

18. Bailey, O. T., and Hass, G. M.: Dural sinus thrombosis in early life. I. The clinical manifestations and extent of brain injury in acute sinus thrombosis, J. Pediat., 11:755, 1937.

19. Bailey, O. T., and Hass, G. M.: Dural sinus thrombosis in early life: recovery from acute thrombosis of the superior longitudinal sinus and its relation to certain acquired cerebral lesions in childhood, Brain, 60:293, 1937.

20. Byers, R. K., and Hass, G. M.: Thrombosis of the dural venous sinuses in infancy and in childhood, Am. J. Dis. Child., 45:1161, 1933.

THE PRESENT STATUS OF THE POLIOMYELITIS PROBLEM*

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It is quite appropriate that poliomyelitis should be a topic for discussion at a meeting of the Louisiana State Medical Society because it was a physician in this state who reported the first epidemic of the disease in this country. The author of this brief report, George Colmer, practiced in the Florida Parishes and resided, at the time of his report, 1841, in Springfield, Livingston Parish.[1, 2]

My report, although somewhat longer, lacks the originality of Colmer's because it is not based on original investigation but rather on a review of recent literature made for the purpose of finding out where we stand in this problem. As time does not permit a complete discussion of so broad a subject, my remarks may be colored by my own interpretation and I shall be forced to disregard a careful balance of both sides of some controversial topics.

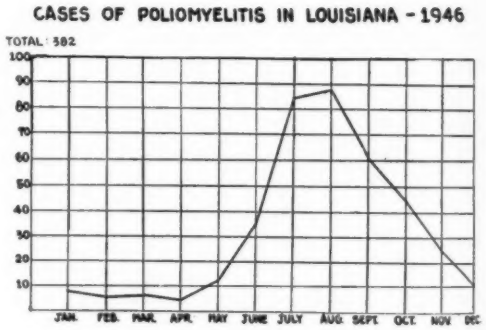
In talking with a group of practicing physicians who have varied and broad interests and are primarily concerned with the more practical aspects of medicine, one is always tempted to emphasize diagnostic and therapeutic procedures. Despite the fact that our knowledge of the disease is increasing rapidly and that investigation will lead eventually to more specific methods of diagnosis and treatment of the acute infection, I should warn you at the beginning that I have little to offer in that regard.

ORGANISM

The virus of poliomyelitis is one of the smallest known and, as is true of filtrable viruses in general, propagates only in living cells. It is, however, resistant to freezing, drying and many protoplasmic poisons and may be preserved in the ice chest, in nervous tissue, for several years. It is neurotropic, in fact "neuronotropic", in that it combines preferentially with the neuron itself rather than the glial elements of the nervous tissues.

EPIDEMIOLOGY

The seasonal incidence of the disease, with prevalence in the summer months, is well known. If it occurred only during that period of the year, study of the epidemiology might be much more simple, but the fact remains that it occurs in endemic form throughout the year. Cases are seen, even in the Northern states, during the winter months. The distribution of reported cases during the 1946 epidemic in Louisiana is shown in figure 1.



LEGEND:

Fig. 1: Monthly incidence of poliomyelitis in Louisiana during 1946.

The mode of transmission has been studied intensively in recent years and many facts, which may lead to methods of prevention, are known. Presence of the virus in the pharyngeal mucus as well as the pharyngeal tissues of persons ill with the disease has quite naturally led to the belief that droplet dispersion may play a role in transmission. It is also known that the virus

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is usually demonstrable in the intestinal walls and feces of infected persons as well as healthy contacts.[3] Trask, Paul and others[4, 5] have demonstrated the virus in urban sewage during epidemic periods. It has not been found in water supplies. It has also been detected in the bodies of various species of flies trapped in epidemic areas.[6, 7, 8]

The bulk of evidence now favors fecal contamination as an important method of transmission. With the seasonal incidence of the disease parallel to that of the fly population, one is tempted to conclude that flies may play the same role in mechanical transmission of the virus that they do in typhoid fever or dysentery. More information must be gathered, however, before precise interpretations can be made. For instance, it will be necessary to know the survival times of the virus outside of the body in feces, mucus and food and the minimal infective dose for man.

PATHWAYS OF INVASION

It has been quite definitely established that having gained entrance to the animal body, the virus is transmitted along healthy nerve fibers. This has been demonstrated repeatedly by the following rather simple experiment. If a sciatic nerve is sectioned and the virus suspension applied to the proximal cut end, poliomyelitis invariably results after an incubation period of four to six days. If, on the other hand, the nerve is cut or even frozen, proximal to the point of application of the virus, no infection results.

In the macacus rhesus monkey, which was practically the only animal used for this work prior to 1938, the disease can be produced readily by application of the virus to the nasopharynx but not to other mucous membranes or the skin. If the olfactory nerves of this animal are sectioned, invasion does not take place no matter where the virus is applied externally. Thus, as long as our observations were confined to the macacus rhesus, the problem was simple. The portal of entry was the nasopharynx.

It has become increasingly evident that conclusions regarding the portal of entry in man cannot be based on observations in the rhesus monkey for many reasons, foremost among which is the fact that the virus is not usually found in the stools of infected animals of that species and that entry via the gastrointestinal tract may be barred by intestinal secretions or some other mechanism.

There are many reasons to believe that the portals of entry in the chimpanzee may be more similar to those in man. As is also true in man, the virus can be recovered from the stools, and two young chimpanzees in a zoo are reported to have contracted the disease spontaneously during an epidemic.[9]

In one series of experiments by Howe and Bodian,[10] six chimpanzees were inoculated in pairs with human stools containing poliomyelitis virus. Two animals received the inoculum intranasally, two intraorally and two by stomach tube. In each of the last two pairs, one animal had been subjected to bilateral olfactory tract section. All of the animals contracted poliomyelitis. The entire brain stem and considerable portions of the cortex, spinal cord and peripheral nervous system were studied by serial section. Lesions of the olfactory bulbs and secondary olfactory nuclei were present only in the animals receiving intranasal inoculations. There was good correlation between the development of bulbar paralysis and intraoral inoculation. In the animals receiving stomach tube inoculations, there was histologic evidence that the virus had reached the brain from the spinal cord. Another animal, not included in this series, was killed before paralysis and showed lesions of the coeliac and sympathetic chain ganglia but none of the central nervous system.

Studies on the portal of entry in man must be approached more indirectly and are therefore less conclusive than in other animals. Two methods have been used: pathologic analysis of the distribution of lesions and virus assay of tissues. Neither of these methods is entirely satisfactory because at the time of death, the virus is widely disseminated. Furthermore, histologic methods of tracing the spread of infection are available only for nervous tissues. His-

tologic findings show that the olfactory bulbs are capable of reacting to the virus but indicate that the olfactory portal is not involved in most cases.

Many facts point to the alimentary tract as a focus of virus proliferation and the spinal cord as the primary site of invasion of the central nervous system in man. Critical evidence of the abdominal sympathetic system as a route to the spinal cord has not been produced but it can be strongly suspected on the basis of the demonstration of both the virus and histological lesions in the coeliac ganglion of chimpanzee and man.

SYMPTOMATOLOGY AND PATHOLOGY

As this group of practicing physicians is no doubt familiar with the symptoms of the disease, I shall not take the time necessary to recite them from the textbook but shall attempt rather to review the course of the disease in relation to our knowledge of the underlying pathology and immunology.

We have reviewed the mode of transmission and the evidence available regarding invasion of the nervous system. The virus having entered the nervous system, one of three courses may follow: The disease may be arrested in the "abortive" or "non-paralytic" stage, paralysis with recovery may take place or permanent paralysis may result.

The first symptoms of the disease are fever, headache, pharyngitis, vomiting and diarrhea. These are followed in one to three days by irritability or drowsiness, muscle tenderness, stiffness of the neck, rigidity of the spine and positive Kernig sign. These symptoms usually appear seven to 14 days after exposure. Examination of the spinal fluid at that time usually reveals 50-100 cells, although the count may be higher or lower. Polymorphonuclear leucocytes may be present during the first few days but lymphocytes predominate. The globulin content of the spinal fluid is normal or slightly increased at first but gradually increases. From the clinical standpoint, the disease may not, and probably in the majority of cases does not, progress beyond that stage. The existence of non-paralytic cases in man is, of course, difficult to establish. In the experimental animal, however, it can be confirmed histologically and by virus assay.

When virus reaches the central nervous system, one of several sequels is possible. If the dose is subinfective for the cells near the site of entry, the virus may be sterilized there. If the dose is somewhat larger or the tissues more susceptible, a local pathological response to invasion and injury at the site of entry may occur, followed by sterilization before the virus can multiply and spread to other susceptible centers, or there is multiplication and spread to other susceptible centers.[11] Failure of multiplication and spread and injury to a comparatively few cells probably accounts for the majority of non-paralytic cases. The absence of demonstrable paralysis in many cases is probably due to the fact that the distribution of destroyed motor neurons is too scattered to involve a single functional muscle group sufficiently to produce clinically evident loss of function. In such cases, one may find all the anterior horn cells destroyed in one miscroscopic section and another section a millimeter away unaffected.

It is generally agreed that a large portion of cases never progress beyond the non-paralytic stage. The extent of invasion beyond this point is dependent upon the balance between virulence of the organism and susceptibility of the host. When paralysis occurs, weakness is usually observed and reflexes diminished by the third day after onset of symptoms. The maximum degree of paralysis is reached in a few hours to two or three days and the fever usually falls by lysis in four or five days.

Of those in whom a definite weakness can be demonstrated, approximately 25 per cent show complete clinical recovery, five to ten per cent die, and the remainder have some degree of residual paralysis.

Time does not permit a careful discussion of the morphologic changes which take place in the nervous system as the disease advances. Briefly, the major lesions consist of degeneration of the anterior horn cells of the spinal cord and motor nuclei of

the cranial nerves and, in the acute state, widespread inflammation throughout the nervous system. The virus migrates along the living axon and is distributed widely throughout the central nervous system. How it can pass through certain neurons without producing injury remains a mystery but it seems likely that chemical differences exist and that the type of cell-selectivity found in other virus diseases may apply.

PREVENTION

The possibilities of establishing effective control of the disease fall into three categories: prevention from exposure, passive immunization and active immunization.

Our knowledge of the transmission of the disease is so limited that few definite recommendations can be made on the control of exposure but I would warn against questionable measures which encourage public panic.

On the basis of evidence that the virus may be recovered from the pharynx, protection of children from exposure to infected persons and from crowds during epidemics may be justified but it seems unlikely that droplet dissemination will prove to be the primary method of transmission. Control of spread of the virus from one intestinal tract to another may be a more efficient approach. As in typhoid, healthy carriers have been recognized and patients who have recovered from either the paralytic or non-paralytic types of disease carry the virus in their intestinal tracts for various periods. It presents a much more difficult problem than typhoid, however, because the virus can be identified only by animal assay and that is too expensive and time-consuming a procedure to be carried out on a large scale. Our current methods and duration of isolation are arbitrary and not based on scientific evidence.

Attempts to control exposure by nasal sprays have not proved efficacious. They were based on the assumption that the primary portal of entry in man, as in the rhesus monkey, is through the olfactory tract and that assumption is no longer valid.

Considerable attention has been given recently to the problem of tonsillectomy in relation to poliomyelitis. Children who develop the disease shortly after tonsillectomy are more likely to have it in the fatal bulbar form and the incidence may be higher among this group. Until better evidence that it is a safe procedure is available, it would seem advisable to avoid tonsillectomy during epidemics. This does not mean that the operation should not be performed during the summer months in years or areas in which the prevalence of the disease is not increased.

On general principles, it does not seem likely that passive immunization offers much hope as a preventive measure. The best we can hope for is that a concentration of antibodies, such as is found in human gamma globulin, may offer temporary protection to children known to have been exposed, if given before the virus enters the body. The evidence at present is discouraging.

Active immunization is a topic of much more lively interest. It offers the most hopeful approach to the control problem that we know at the moment but the ideal vaccine has not yet been discovered. The disappointing results which followed the use of killed and chemically attenuated viruses are well known. When the killed virus was used, apparently no protection resulted. When the chemically attenuated virus was used, it was not sufficiently attenuated and children developed the disease. Much work has been done recently on mouse-adapted strains and on attenuation of the virus by irradiation and other physical means. One of the limitations in all of this work is the fact that the presence of neutralizing antibodies in the circulating blood may not guarantee protection of the neuron. Neutralizing antibodies have been demonstrated very early in the disease. This suggests that they were present at the time of exposure and did not serve a protective function. Furthermore, second attacks have been reported, although it is quite possible that both attacks were not caused by the same strain of virus.

THERAPY

Regarding specific therapy in the acute phase of the disease, there are few encour-

aging leads. Numerous drugs, including the sulfonamides and penicillin, have been tried and have failed. Fifteen years ago it was thought that convalescent human serum was the answer but most investigators now agree that it is of no value.

Use of the concentrated gamma globulin fraction of pooled adult human serum in preparalytic poliomyelitis has been the subject of considerable active research recently. Neutralizing antibodies against the Lansing strain of poliomyelitis are said to be present in this fraction at an average concentration 18 to 25 times as high as that in the corresponding whole plasma. Bahlke and Perkins[12] conducted a very critical investigation of the efficacy of gamma globulin during the 1944 epidemic in New York State. Their conclusions are, "In a series of 111 patients with preparalytic poliomyelitis observed for approximately six months after onset, no benefit is detectable when 56 of them who had received large doses of gamma globulin intramuscularly are compared with 55 alternate, untreated controls."

I shall not enter the controversy surrounding the orthopedic treatment of the disease once paralysis has developed. In general it appears that complete immobilization of extremities may be disadvantageous in many cases because it may lead to atrophy and increased loss of function. Application of heat relieves pain and when combined with active physical therapy, diminishes the extent of paralysis and deformity. Maintenance of centers where the disease can be treated actively in the early stages of paralysis is certainly justified.

The ramifications of this subject have made it necessary for me to review hastily a large amount of material. I have tried to indicate the directions in which progress may be expected. Despite the enormous amount of effort expended in the study of this disease in recent years, we are far from the goal.

DISCUSSION

Dr. Peters: What do you think about the CO_2 treatment?

Dr. Lippard: I have had no experience with it and I do not understand the rationale underlying its use.

Dr. Levy (Baton Rouge): What has been your experience at Charity Hospital with those patients who have received sulfa drugs in the prodromal stage? Is it true that the paralysis has been worse?

Dr. Lippard: It is my impression from the literature that they have no effect on the disease one way or another. That would seem logical because the sulfa drugs are, in general, not antiviral in their action.

REFERENCES

1. Colmer, G.: Paralysis in teething children, Am. J. Med. Sc., 5:248, 1843.

2. Casey, A. E., and Hidden, E. H.: George Colmer and the epidemiology of poliomyelitis, South. M. J., 37:471, 1944.

3. Trask, J. D., Vignec, A. J., and Paul, J. R.: Poliomyelitis virus in human stools, J. A. M. A., 111:6, 1938.

4. Paul, J. R., Trask, J. D., and Culotta, C. S.: Poliomyelitic virus in sewage, Science, 90:258, 1939.

5. Paul, J. R., Trask, J. D., and Gard, S.: Poliomyelitic virus in urban sewage, J. Exper. Med., 71:765, 1940.

6. Paul, J. R., and others: Detection of poliomyelitis virus in flies, Science, 94:395, 1941.

7. Trask, J. D., Paul, J. R., and Melnick, J. L.: Detection of poliomyelitis virus in flies collected during epidemics of poliomyelitis: methods, results, and types of flies involved, J. Exper. Med., 77:531, 1943.

8. Sabin, A. B., and Ward, R.: Flies as carriers of poliomyelitis virus in urban epidemics, Science, 94:590, 1941.

9. Muller, W.: Spontane poliomyelitis beim schimpansen, Monatschr. f. Kinderh., 63-134, 1935.

10. Howe, H. A., and Bodian, D.: Neural Mechanisms in Poliomyelitis, The Commonwealth Fund, New York, 1942, pp. 97-124.

11. Sabin, A. B.: Bela Schick lecture; studies on natural history of poliomyelitis, J. Mt. Sinai Hosp., 11:185, 1944.

12. Bahlke, A. M., and Perkins, J. E.: Treatment of preparalytic poliomyelitis with gamma globulin, J. A. M. A., 129:1146, 1945.

13. (Only those articles to which direct reference is made are listed. For a general bibliography on poliomyelitis, the reader is referred to Fishbein, M., Hektoen, L., and Salmonsen, E. M.: A Bibliography of Infantile Paralysis, 1789-1944, J. B. Lippincott Co., Philadelphia, 1946).

INTRAMEDULLARY INFUSIONS IN INFANTS*

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NEW ORLEANS

More than 200 patients admitted to the pediatric wards of Charity Hospital in New Orleans have received fluids by intramedullary route since December, 1945. At that

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time a simplified procedure was established by the Tulane Pediatric Division; because no complications have occurred, our experiences and this technic deserve discussion.

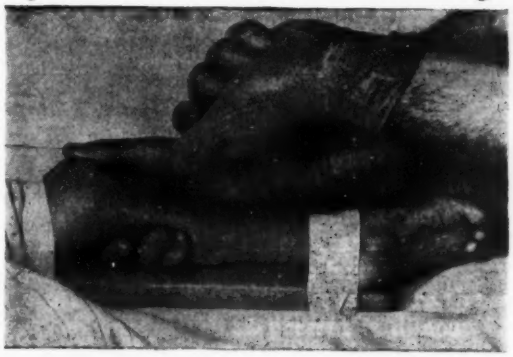
In 1932 Drinker and associates[1] and Doan[2] independently suggested bone marrow as a route for administration of fluids. Tocantins and co-workers[3, 4, 5] enlarged on earlier work in this field, and published data regarding indications, rate of absorption, and complications. Since 1942 several reports from abroad have emphasized the usefulness of this route for fluid injections in properly selected patients. Heinild, Sondergaard and Tudvad[6] recently reported experiences with 1000 bone marrow infusions in six different Danish hospitals. They condemned long-continued infusions and intra-osseous injection of hypertonic or irritating fluids. They recommended the multiple syringe, "push" infusion as the most satisfactory method.

TECHNIC

Many methods of administering fluids into the medullary cavity have been described, some calling for elaborate needles or trephines, others requiring only minimal simple equipment. In our experience an 18 gauge, stainless steel needle with a well-fitting stylet is adequate; strength and a keen cutting edge are essential. For older infants a 16 or 17 gauge needle of the same quality may be employed.

The patient is prepared in the following manner: The lower extremity is fixed to a padded board so that the assistant operator's hand may support the knee. The upper two-thirds of the tibia is cleansed, then prepared with iodine or merthiolate followed by alcohol, as for any surgical procedure; sterile technic is used throughout. One c.c. of normal saline containing 10,000 units of penicillin is mixed with 0.25 c.c. of 1 per cent solution of procaine hydrochloride. A site is chosen in the mid-portion of the upper third of the medial (flat) tibial surface. Particular care is used to avoid proximity to the epiphyseal line. The penicillin-procaine solution is then infiltrated down to the periosteum.

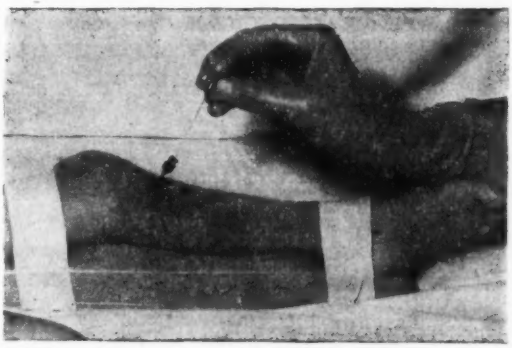
A 5 c.c. Luer-lok syringe is attached to the needle selected, and a small towel is wrapped around the syringe; this then serves as a handle. The needle is directed toward the knee not quite perpendicularly to the long axis of the tibia, and pressure is exerted while employing a slightly drilling motion. One hand supports the tibia



LEGEND:

Fig. 1: Puncture of tibial medullary cavity with 17 gauge needle with 5 c.c. Luer-lok syringe as handle. Note support of patient's extremity as pressure is applied.

during this maneuver (see fig. 1). Entry into the marrow cavity is easily appreciated by a sudden "give" or reduction in resistance. The bevel of the needle is directed cephalad, the syringe is removed, and a stylet introduced to be sure the lumen is



LEGEND:

Fig. 2: Introduction of stylet to insure patency of needle lumen. If bony core is present, a few thrusts will dislodge it.

clear (see fig. 2). The ease with which the bony cortex may be punctured is dependent upon skill of the operator, site chosen, age of

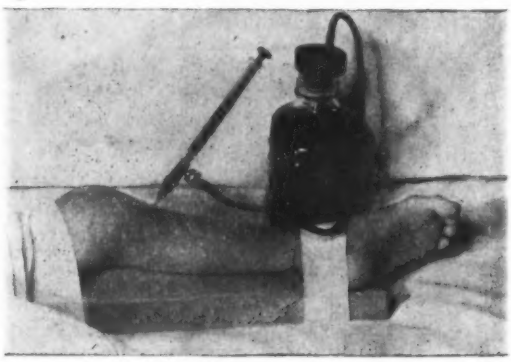
the patient, and perhaps certain deficiency syndromes. Despite these considerations, however, one often encounters extreme and unexplained variations in thickness, resistance, and brittleness of bone, and consequent minor technical difficulties which are easily overcome with experience. A few c.c. of saline is injected, and if this is blood-tinged when aspirated, the syringe is removed and the special infusion set is attached.

A year ago it was our policy to employ the multiple syringe method, so uniformly recommended throughout the literature. We now wish to introduce an infusion set, which, to the best of our knowledge, has not hitherto been described for this purpose (see fig. 3 and 4). This infusion set has many advantages. Once attached to the marrow needle, the system is closed. Because the valve is automatic and light in weight, the possibility of needle movement is minimized and likelihood of seepage into surrounding tissue reduced; the latter is a frequent complication of the multiple syringe method. Extreme simplicity of operation has increased its popularity. Almost any small syringe may be employed with this set, but for convenience, and to reduce potential pressure-pain, we ordinarily use a 5 c.c., 2 c.c., or even a tuberculin syringe for infants. Such a set is now standard equipment on our service, and will be described in detail elsewhere.

If a continuous infusion is desired, the marrow needle is fixed in place with flamed adhesive and sterile gauze (see fig. 5). We have encountered no complications even when infusions have remained for as long as 72 hours, though usually they are not allowed to run for more than 12 to 24 hours. If additional parenteral fluids are required after this time, the preferred intravenous route is usually again available. Bone marrow infusions may be repeated, however, as often as necessary simply by varying sites of injection, but the same bone should

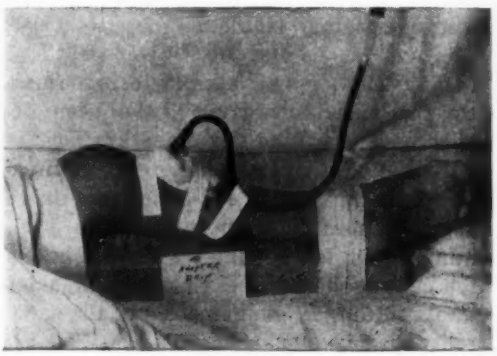
LEGEND:

Fig. 3: Two-way automatic valve with small syringe attached ready for attachment to marrow needle. Note very thin plasma tubing.



LEGEND:

Fig. 4: Automatic valve attached to marrow needle. Mantoux syringe attached is popular because of extremely light weight and because only slight pressure is needed to force fluid.



LEGEND:

Fig. 5: Light weight BD Luer-lok adapter attached to marrow needle for marrow drip. Blood and plasma are difficult to administer by gravity alone, but less viscous fluids can be administered by this method.

not be punctured again until at least 24 hours have passed.

Before the needle is removed, 10,000 units of penicillin in normal saline is injected partly into the medullary cavity and partly along the course of the withdrawing needle. We have done this routinely.

We do not recommend the intramedullary over the intravenous route, but use it rather as an alternative when veins are inaccessible. Just when patients most urgently need corrective fluid therapy, valuable time is often lost by attempting to enter collapsed or very small veins. Added trauma incident to such well-meaning efforts and the extra manipulations they entail, certainly appear further to depress an already debilitated patient.

REPORT OF A CASE

A recent illustrative incident occurred when a malnourished infant with a severe acute nutritional disturbance was admitted. Comatose, he exhibited all the classical features of marked dehydration and acidosis; the carbon dioxide combining power was 7 volumes per cent. There was twitching of the extremities and no discernible reaction to painful stimuli. Death appeared imminent. Fourteen venepunctures and two cutdown procedures had been attempted. Appropriate alkaline and fluid therapy was then administered by rapid, forced injection into the tibial marrow. Response was gratifying, and the infant was crying vigorously within an hour. Usual supportive and dietary measures were then employed, and a transfusion was given into the same needle six hours later. Uneventful recovery ensued.

We see many such emergencies among severely dehydrated infants brought to Charity Hospital, and it has been our policy to administer fluids immediately by the most expeditious route. Injudicious delay in administration of corrective solutions may be hazardous and may further accentuate existing water and electrolyte deficits. The rapid and almost foolproof technic for marrow infusions can be mastered by witnessing the procedure two or three times, whereas many intravenous technics useful for infants require time and skill and therefore often present insurmountable difficulties to the occasional or inexperienced operator.

We have successfully completed more than 200 intraosseous infusions in infants and small children ranging from young prematures weighing as little as 964 grams (2 pounds, 2 ounces), and there have been no complications or sequelae. One patient received fourteen such infusions during a period of seven weeks. The puncture technic is not ordinarily used or recommended for children over the age of three, though there is no actual contraindication when an emergency exists beyond this age.

Pain, so obviously related to the pressure used for marrow infusions is ordinarily reduced or eliminated when the rate and pressure are reduced. Pain thus serves as a useful guide for controlling speed of fluid administration.

Tocantins and O'Neill[4] and Quilligan and Turkel[7] found frequent warnings in the literature two decades ago regarding local complications following prolonged administration of fluids into any single vein, and many incriminations of this now-common route also warn us of such dangers following cutdown procedures. Complications after intramedullary infusions into long bones, such as osteomyelitis, pressure occlusion of vessels, cellulitis, and subcutaneous abscesses, have been reported, though none of these are common. Quilligan and Turkel[7] found only 12 such among 1049 patients who had received fluids by intramedullary route. Five of these had mediastinitis, following sternal marrow punctures; five others had osteomyelitis of long bones.

We have not used the sternal route, and to reduce the possibility of infection, penicillin was infiltrated at the site of puncture before the needle was inserted, and again as it was withdrawn.

Queries have been received regarding rates of absorption for various types of fluids. While there is no doubt that fluids can be given more rapidly intravenously, rates attained in intramedullary infusions are ordinarily adequate. We give blood, plasma, or any of the physiologic repair solutions freely, employing usual dosage rules. Meyer and Perlmutter[8] found that the circulation time in human subjects measured from corresponding intravenous

and intramedullary sites was essentially similar in 21 subjects. They concluded that the intimate relationship between the general circulation and the medullary cavities warrants their use when venous channels are not readily accessible.

SUMMARY

1. Intramedullary infusions should be reserved for patients whose veins are inaccessible.

2. The puncture principle, employing a short 16-18 gauge stainless steel needle is described.

3. Viscous fluids, such as blood and plasma, may not drip in easily by gravity alone; forced pressure injection is often necessary.

4. A two-way automatic valve with syringe pump seems more satisfactory than multiple syringe or continuous infusion methods.

5. All common infusion fluids may be administered by this technic at a satisfactory rate. Irritating solutions should not be used, lest tissue injury result.

6. We believe that absence of complications among these infants can best be attributed to careful technic, avoidance of the sternal route, use of penicillin at the site of marrow puncture, and to the closed system we ordinarily employ.

DISCUSSION

Question (by student): Does seepage occur at the site? What bone other than the tibia do you use?

Dr. Sutton: We use the tibia in 95 per cent of the cases; in other cases the femur. The femur is good in permaturees.

Question (by student): Do you resterilize the needle and use it over again or use a new needle each time?

Dr. Sutton: I think the cost is so little that it justifies the use of a new needle.

REFERENCES

1. Drinker, C. K., Drinker, K. R., and Lund, C. C.: The circulation in mammalian bone marrow, Am. J. Physiol., 62:1, 1922.

2. Doan, C. E.: The circulation of the bone marrow Contrib. Embryol., 14:27, 1922.

3. Tocantins, L. M.: Rapid absorption of substances injected into the bone marrow, Proc. Soc. Exper. Biol. & Med., 45:292, 1940.

4. Tocantins, L. M., and O'Neill, J. F.: Infusion of blood and other fluids into the circulation via the bone marrow, Proc. Soc. Exper. Biol. & Med., 45:782, 1940.

5. Tocantins, L. M., O'Neill, J. F., and Price, A. H.: Infusions of blood and other fluids via the bone marrow in traumatic shock and other forms of peripheral circulatory failure, Ann. Surg., 114:1085, 1941.

6. Heinild, S., Sondergaard, T., and Tudvad, F.: Bone marrow infusion in childhood, J. Ped., 30:400, 1947.

7. Quilligan, J. J., Jr., and Turkel, Henry: Bone marrow infusion and its complications, Am. J. Dis. Child., 71:461, 1946.

8. Meyer, L. H., and Perlmutter, M.: Absorption rate from bone marrow, Am. J. Med. Sc., 205:187, 1943.

HUMAN INFECTION WITH VIRUS OF EQUINE ENCEPHALOMYELITIS, EASTERN TYPE, IN LOUISIANA

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G. PEYTON KELLEY, M. D.††
AND
MORRIS F. SHAFFER, D. Phil.†
NEW ORLEANS

Present knowledge of human infections with the virus of equine encephalomyelitis, Eastern type, rests largely upon studies performed on the 34 or more cases of the 1938 epidemic in Massachusetts, at least 24 of which were in children under ten years of age. Farber, Hill, Connerly, and Dingle[1] examined eight of these cases occurring in infants and children, reviewing the clinical features, diagnosis, treatment, pathologic and laboratory findings. Seven of the eight cases similarly reported by Wesselhoeft, Smith, and Branch[2] fell into roughly the same age group. The virus was isolated repeatedly at that time by Webster and Wright[3] as well as by Fothergill and associates,[4] but it does not appear to have been recovered from any subsequent instance of the disease in man. In 1941 Hammon[5] reported one case in an 8 year old girl, and in 1942 Bohls and Irons[6] recorded two probable infections in patients of unstated age. These three cases, occurring in Texas, were non-fatal; the diagnosis was based on clinical evidence supported by demonstration of specific virus-neutralizing properties in the patients' sera. To augment existing data, the following description of a recent fatal infection in a 6 year old girl from Louisiana is presented; isolation and identification of the virus are likewise reported.

CASE REPORT

Clinical History: On the morning of August 3, 1946, B. H., a 6 year old white girl, resident of

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Houma, La., awoke complaining of headache. Her mother thought she had fever. She vomited twice during the afternoon. Following a short generalized convulsion she was taken to a local hospital where her temperature was found to be 101° F. No laboratory tests were performed, and after one injection of penicillin she was sent home. The patient played in her bed that evening, feeling better, but upon the reappearance of fever, headache, and vomiting next morning she was referred to the Charity Hospital of Louisiana at New Orleans.

Physical examination on admission disclosed a well-developed, well-nourished child who appeared moderately ill. She was stuporous but responded to stimuli and was cooperative when aroused. Kernig's and Brudzinski's signs were positive, hyperactive tendon reflexes were noted, and the pharynx was moderately injected. Temperature was 103.8° F., blood pressure 120/80 mm. Hg. Petechiae appeared on the abdomen within a few hours.

The initial hematologic examination showed 19,000 leukocytes per cu. mm. of which 88 per cent were polymorphonuclears, 12 per cent lymphocytes; the hematocrit was 34 per cent. During hospitalization two specimens of urine were examined and found normal; two blood cultures and a Mantoux test (0.1 mgm. O.T.) proved negative. The spinal fluid findings in four lumbar punctures are summarized in table 1 and are considered typical for viral encephalitis, though perhaps the initial content of leukocytes was higher than usual; smears and cultures from each specimen of fluid were reported as bacteriologically negative.

TABLE 1

SUMMARY OF FINDINGS ON EXAMINATIONS OF SPINAL FLUID FROM PATIENT B.H.

Date	Pressure	Leukocytes: Total	Leukocytes: Per cent Pmn.	Pandy	Protein Mg./100 c.c.	Sugar Mg./100 c.c.	Chlorides Mg./100 c.c.
Aug. 4	250	1600	100	+	45	64	715
" 5	.....	760	90	+	55	60	585
" 6	460	510	82	+	93	84	644
" 6	.....	26(?)	44	0	....	48	667

By 3:00 a. m. of August 5, recurrent convulsions and muscular twitchings had developed; the patient was unconscious, remaining so until death. After the first convulsion, plantar reflexes became extensor. Nystagmus and mild bilateral papilledema were noted. On August 6, the patient's condition deteriorated progressively. Mucus accumulated in the throat, and severe bilateral papilledema developed rapidly. The next day, August 7, she exhibited evidences of peripheral vascular collapse. Her temperature ranged between 104° and 106° F., blood pressure dropped to 85/60 mm. Hg. At 5:00 p. m., bulbar symptoms increased, respirations became shallow, the face was flushed, and the patient died shortly thereafter.

Treatment was based on an initial presumptive diagnosis of pyogenic meningitis and included vigorous supportive measures. Sulfadiazine was given in dosage of 0.2-0.3 Gm./Kg. daily by hypodermoclysis. Ten thousand units of penicillin was administered intrathecally at the time of the initial spinal puncture; thereafter large amounts were given by intravenous, subcutaneous, and intrathecal routes. Repeated small transfusions of whole blood were given by continuous intravenous drip. Dehydration was controlled by parenteral administration of fluids to assure a total daily intake of 110 c.c./Kg. Concentrated plasma and 50 per cent glucose were given in an attempt to combat cerebral edema. Barbiturates were used for sedation. During the last two days of illness inhalations of 5 per cent carbon dioxide in oxygen were used in an effort to liquefy secretions. Adrenal cortical extract had no remarkable effect in altering the features of terminal collapse.

The final clinical diagnosis was viral encephalitis, type undetermined. Clinical and epidemiologic considerations justified suspicion of infection with the virus of equine encephalomyelitis.

Postmortem Observations:* Postmortem examination was restricted to the head. An extremely edematous brain weighing 1500 gm. was removed. No gross evidences of meningitis were found, but hyperemia and small hemorrhages were noted about the base. After removal of material for virus study, the remainder of the brain was fixed in formalin. Sections were later taken from the following regions and stained with hematoxylin-eosin: frontal, temporal and occipital lobes; wall of the anterior and posterior horns of the lateral ventricles; thalamus; mesencephalon; wall of the fourth ventricle; basal ganglia; cerebellum. Microscopic examination** revealed similar changes in all samples of the brain. There were found in both the grey and white matter diffuse and focal areas of neutrophilic infiltration, the latter amounting sometimes to small abscesses. Accumulations of neutrophils and areas of demyelinization were observed around many of the blood vessels; demyelinization was noted especially at junctions of grey and white matter. The meninges exhibited small hemorrhages and moderate-to-severe infiltration of neutrophils, lymphocytes and macrophages.

Virus Studies: Several small portions of brain taken at autopsy shortly after death on August 7, 1946 were placed in glycerine and stored at 4° C.

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*We are indebted to Dr. E. S. Moss, Director, Department of Pathology, Charity Hospital, for permission to study specimens obtained at autopsy.

**We are indebted to Dr. James B. Arey for examination of the sections.

On August 10, 1946 approximately one gram of this material was ground in a mortar and emulsified in broth. Groups of adult white mice were then inoculated by each of the following routes: intracerebral, intraperitoneal, intranasal, and subcutaneous. Six mice inoculated intracerebrally died within two to five days, and one mouse inoculated intraperitoneally died on the fifth day, each with signs characteristic of encephalitis in these animals.

The brains of the dead mice were emulsified and inoculated into other mice by the intracerebral route. This emulsion and similar preparations from later passages were found to be bacteriologically sterile; they contained an agent which passed through a Seitz EK filter disc and regularly killed white mice in 36 to 72 hours. In chick embryos, infection was lethal within 24 hours. The short incubation period in experimental animals suggested virus of equine encephalomyelitis; accordingly, an attempt was made to compare the agent isolated with known Eastern and Western types of equine encephalomyelitis virus‡ by means of cross-immunization and serum-protection tests.

Thirty-six mice were given a series of intraperitoneal injections of formalinized chick embryo vaccine prepared from the unknown virus. Groups were then challenged with varying dilutions of known strains of Eastern and Western types of equine virus. As shown in table 2, immunization with the agent under study protected mice against as many as 10,000 MLD's of Eastern type virus, but gave no significant protection against the Western type.

Three consecutive serum protection tests, performed according to the intraperitoneal technic of Olitsky and Harford† showed that mice could be protected against at least 1000 MLD's of the newly isolated virus by guinea pig antiserum to Eastern type equine virus†, whereas antiserum to the Western type of equine virus was without prophylactic effect. The results of one such experiment are summarized in table 3.

TABLE 3

RESULTS OF PROTECTION TEST WITH EQUINE ENCEPHALOMYELITIS ANTISERA AND VIRUS FROM PATIENT B. H.

Serum used for Protection	Final Dilution of Virus Injected			
	10^{-4}	10^{-5}	10^{-6}	10^{-7}
Anti-Eastern Type	3/3*	3/3	3/3	3/3
Anti-Western Type	.....	0/3	0/3	0/3
Normal Guinea Pig Serum	.....	0/3	0/3	0/3

*Numerator represents number of survivors after five days of infection; denominator represents number of mice challenged.

It was concluded that the agent isolated from the brain of patient B. H. was virus of equine encephalomyelitis, Eastern type.

DISCUSSION

On the day that B. H. was admitted to Charity Hospital an elderly woman, also of Houma, La., was admitted to a different service in the same institution. Her disease coincided with that of B. H. in time of onset, symptoms, and clinical course, terminating fatally on the same day. No autopsy was performed, and unfortunately the resemblance to our patient was not appreciated until it was too late to secure blood or spinal fluid for study. So far as we have been able to determine, there were no previous illnesses of the same nature reported from that region, and none had developed up to August, 1947.

The evidence of limited outbreaks in Texas and Louisiana suggests the possibility that cases of human infection with this virus may be more frequent than published reports would indicate. Since 1940, 28 human cases diagnosed as encephalitis, of unproved etiology, have been reported to

TABLE 2

RESULTS OF CHALLENGE INOCULATIONS IN MICE IMMUNIZED WITH VACCINE FROM VIRUS ISOLATED FROM PATIENT B.H.

Group	Type of Equine Encephalomyelitis Virus Used for Challenge	Dilution of Virus Injected					
		10^{-3}	10^{-4}	10^{-5}	10^{-6}	10^{-7}	10^{-8}
Immunized	Eastern E. E. Virus	0/3*	3/5	5/5	4/5	.....	.....
	Western E. E. Virus	.....	0/3	0/5	0/5	1/5	.....
Controls	Eastern E. E. Virus	.....	.....	0/2	0/2	0/2	0/2
	Western E. E. Virus	.....	.....	0/2	0/2	0/2	0/2

*Numerator represents survivors after five days of infection with virus used for challenge; denominator represents number of mice challenged.

‡The strains employed were supplied by Dr. W. McD. Hammon of the G. W. Hooper Foundation, San Francisco, California.

†Antiserum was sent by Dr. H. Koprowski, Lederle Laboratories, Pearl River, N. Y.

the Louisiana State Health Officer.* That illnesses diagnosed as postinfection encephalitis are not free from suspicion as possible instances of infection with the virus of equine encephalomyelitis is demonstrated by the observation in Massachusetts that some of the proved cases of infection with equine virus occurred in children convalescent from whooping cough.[2]

Our data concerning the disease in horses are inadequate, but there was certainly no large epizoötic during 1946. The virus was recovered from horses in Louisiana in 1944,[8] and many of the potential reservoirs and vectors which have been implicated in studies of the transmission of this infection are found here in abundance. A large outbreak of encephalomyelitis in horses and mules occurred in Louisiana in the summer of 1947, and several human patients suspected of suffering from this infection have been admitted to Charity Hospital. Laboratory studies of these cases are now in progress.

With present methods, a diagnosis of encephalitis due to Eastern equine virus could rarely be established sufficiently early to benefit the patient even if effective means of therapy were known. No distinctive syndrome is yet apparent in the variety of clinical manifestations found among the relatively few examples of this disease previously described in man. As noted above, the early symptoms and relative purulence of the spinal fluid in the patient described here suggested acute bacterial meningitis. The negative smears and cultures of spinal fluid and the sterile cultures of blood did not discourage this view, since it was known that the patient had received penicillin before specimens were collected. The possibility of a viral etiology required consideration when the level of cerebrospinal fluid sugar was found to be normal and when noticeable clearing of the spinal fluid occurred in conjunction with progressively more severe disease.

SUMMARY

1. The clinical and pathologic observations in a case of fatal encephalomyelitis in a 6 year old child from Louisiana are described. Virus of equine encephalomyelitis, Eastern type, was isolated from portions of the brain taken at autopsy. Identification of the virus was established by means of cross-immunization and serum protection tests in mice.

2. Despite the widespread distribution of Eastern type equine encephalomyelitis virus among animals in the United States, this would appear to be the first human case reported since the 1938 outbreak in Massachusetts from which virus has been isolated.

Acknowledgement: The continued interest and helpful suggestions of Dr. Ralph V. Platou are much appreciated by the authors.

REFERENCES

1. Farber, S. Hill, A., Connerly, M. L., and Dingle, J. H.: Encephalitis in infants and children caused by the virus of the eastern variety of equine encephalitis, J. A. M. A., 114:1725, 1940.

2. Wesselhoeft, C., Smith, E. D,, and Branch, C. F.: Human encephalitis; eight fatal cases with four due to the virus of equine encephalomyelitis, J. A. M. A., 111:1735, 1938.

3. Webster, L. T.. and Wright, F. H.: Recovery of eastern equine encephalomyelitis virus from brain tissue of human cases of encephalitis in Massachusetts, Science, 88:305, 1938.

4. Fothergill, L. D., Dingle, J. H., Farber, S., and Connerly, M. L.: Human encephalitis caused by the virus of the eastern variety of equine encephalomyelitis. New England J. Med., 219:411, 1938.

5. Hammon, W. M.: Encephalitis: Eastern and Western equine and St. Louis types, as observed in 1941 in Washington, Arizona, New Mexico and Texas, J. A. M. A., 121:560, 1943.

6. Bohls, S. W., and Irons, J. V.: Laboratory observations on virus encephalitis, Texas State J. Med., 38:260, 1942.

7. Olitsky, P. K., and Harford, C. G.: Intraperitoneal and intracerebral routes in serum protection tests with the virus of equine encephalomyelitis, J. Exp. Med., 68:173, 1938.

8. Miller, A. W.: Annual Report of the Chief of the Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C., 1945.

*Personal communication from Dr. W. L. Treuting, State Health Officer.

FARBER'S TEST IN THE DIAGNOSIS OF CONGENITAL INTESTINAL ATRESIA

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NEW ORLEANS

In 1933, Farber described a simple means to determine patency of the alimentary canal in the newborn infant by examining

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meconium.[1] In his method, meconium is smeared on a glass slide, stained with gentian violet and examined microscopically for cornified epithelial cells and lanugo hairs.

This test is based on knowledge that the embryonic alimentary lumen, from the pylorus to the ileocecal valve, becomes obliterated between the fifth and tenth fetal weeks by proliferation of endothelium and then is recanalized. Rarely, a portion of the solid stage persists as congenital atresia. While in utero, the fetus swallows amniotic fluid, which contains cornified epithelial cells and lanugo hairs during the last two months of gestation. If normal recanalization of the gut has occurred, such formed elements will be found easily in the meconium of the infant. If, on the other hand, atresia persists, they will be lacking. Farber states in his original communication: "Absence of cornified cells is proof that a point of atresia is somewhere present in the alimentary tract."

Here, apparently, is a test of great simplicity which has not been recognized and used to proper advantage. The purpose of this paper is to indicate that epithelial cells are invariably present in the meconium of normal infants and that the test, when done in cases of suspected atresia, has considerable diagnostic and prognostic value.

One hundred full term normal newborn infants with birth weights ranging from 2.5 to 3.75 kilograms were observed during the neonatal period and their meconium examined for lanugo hairs and cornified epithelial cells. None of these infants developed any symptoms or signs of intestinal obstruction. Meconium from each was taken from the center of the excreted mass, smeared on a glass slide, and allowed to dry. The unstained smear was examined microscopically for lanugo hairs. This preliminary examination yielded positive results in 91 cases. The remaining nine were stained after the method outlined by Farber:[1, 2] the slide was immersed in ether for three to five minutes in order to extract fat and then allowed to dry. Next it was stained with Sterling's gentian violet,[3] washed in water, decolorized with acid alcohol and examined microscopically. In the nine cases in which hairs were not seen on preliminary examination, cornified cells were found readily by this method. The remaining 91 smears were then similarly stained and cornified cells were easily demonstrated to be present in each one. This emphasizes the practical simplicity of the test.

During the period from January 1937 to June 1946, 23 cases of suspected intestinal atresia in the newborn were recorded in this hospital. Farber's test had not been done in any of these. Since June 1946 there have been 24 patients in which the question of patency of the intestinal tract has arisen. Because of increased local interest in Farber's test, it was used in 22 of the latter group.

Comparison between these two groups is of interest. In both periods there was an obvious tendency to rely primarily on radiography as a means of diagnosis. That this faith is not always justified is shown by table 1.

TABLE 1
ACCURACY OF DIAGNOSIS RELATED TO TESTS EMPLOYED

Total Number Patients	Number with Farber's test	Number with Radiography	Number with Correct Diagnosis
Group I (1937-1946) 23 Patients	None	17	7
Group II (1946-1947) 24 Patients	22	15	23

These figures, moreover, demonstrate that when Farber's test was employed and carefully interpreted in relation to other findings, accuracy of diagnosis was greatly increased.

Several individuals from the earlier series deserve mention because they illustrate how Farber's test *might* have yielded information not obtainable in any other fashion.

CASE NO. 1

A white male infant, one day of age, began to vomit after feedings and to have abdominal distention. A barium meal was given, and it was noted that the barium churned up and down in the descending portion of the duodenum before passing. On this basis a preoperative diagnosis was made of partial duodenal obstruction due to adhesive

bands. At operation, malrotation was found together with volvulus of the small gut; there was no infarction. Adhesions around the duodenum were released, malrotation and volvulus were corrected. Four days later the child died; necropsy revealed two atretic areas in the jejunum. Had Farber's test been done, such a situation might have been suspected and the outcome altered.

CASE NO. 2

A colored male infant began to vomit feedings and became distended on the second day of life. Diagnostic workup included plain films of the abdomen, which showed laddering of the gut and gas-filled loops, leading to a diagnosis of jejunal atresia. Slight infection of the umbilicus was noted but was not considered remarkable. At operation generalized peritonitis secondary to omphalitis was discovered, but there was no atresia present. This was confirmed by postmortem examination. Here the diagnosis of atresia could have been ruled out by Farber's test and further consideration of the umbilical infection might have led to the correct conclusion earlier.

CASE NO. 3

A colored female infant at two days of age began to have continued vomiting after feedings, with gastric distention and retention. Stools were found to contain bile. A diagnosis of atresia of the duodenum was made; at operation the atresia was found one centimeter distal to the pylorus. With presence of bile in the stool, a negative Farber's test in this case could have predicted the exact location of this lesion.

CASE NO. 4

A six day old colored male infant was admitted with a history of vomiting every feeding and of having passed no fecal matter since shortly after birth. There was complete gastric retention after four hours. A diagnosis of pyloric stenosis was made; operation was delayed while atropine, thickened feedings and other medical management were tried. The baby died and postmortem examination showed not only hypertrophy of the pylorus but associated duodenal atresia. A negative Farber's test would have predicted the futility of medical management.

During the short span of time covered by the second group of cases the personnel in charge of newborn infants were especially alert for any early symptoms or signs which might point to congenital gut defects. As a result, more abnormalities were suspected than were actually encountered. Because of variable abdominal distention, regurgitation of feedings, or difficulties in nursing, such suspicions were carefully investigated for twelve infants. In each of these, Farber's test was done, hairs and cells were found, and congenital atresia was thereby excluded. Further investigation revealed other causes for the symptoms and operation was not performed. Five patients had multiple obvious external congenital defects at the time of birth and Farber's test was performed to rule out associated atresia before feeding was begun.

In three cases of esophageal atresia with tracheoesophageal fistula, the test proved valuable for excluding associated lower atretic anomalies. In the majority of such infants the esophagus connects with the trachea just distal to the blind esophageal pouch, so that hairs and cells from amniotic fluid easily gain access to the gut. Such a connection is best determined by demonstrating air in the stomach roentgenologically. The presence of air in the stomach in the absence of hairs and cells from the meconium strongly suggests intestinal atresia as an associated anomaly. Hairs and cells were present in the meconium of these three patients and the esophageal operation was undertaken.

Three cases of intestinal atresia were encountered in which hairs and cells were not found in the meconium preoperatively. In one of these, an additional use for Farber's test was found. Serial postoperative examination of meconium stools revealed the presence of cells. Thus, during the immediate postoperative period, when oral feeding of dyes or other marker-substances was undesirable, it was possible to be sure that no areas of atresia had been overlooked and that bowel continuity had been established.

In one infant a clinical diagnosis of sepsis of the newborn was made and Farber's test was omitted (see table 1). At necropsy, in addition to sepsis and peritonitis, congenital malrotation of the gut and a duodenal diaphragm were found. With Farber's test such a condition might have been suspected and successfully attacked surgically.

In the initial group of 23 cases, there was a remarkable preoccupation with radiography as the single most important means of diagnosis. In many published accounts of intestinal atresia, Farber's test is al-

luded to in the discussion but it is infrequently included as a preoperative diagnostic procedure.[4]

CONCLUSIONS

1. Cornified epithelial cells were invariably present in meconium of one hundred normal newborn infants. Lanugo hairs were found in 91 per cent of unstained meconium smears. The presence of lanugo hairs and epithelial cells in meconium is evidence of continuous patency of the intestinal tract.

2. This simple examination (Farber's test) should be carried out routinely within the first 48 hours of life when there is any suspicion of gastrointestinal dysfunction. When the results are correlated with symptoms, physical signs, and roentgenologic findings in a given case, accuracy of diagnosis can be materially increased.

REFERENCES

1. Farber, S.: Congenital atresia of the alimentary tract, J. A. M. A. 100:1753, 1933.
2. Ladd, W. E., and Gross, R. E.: Abdominal Surgery in Infancy and Childhood, W. B. Saunders Company, Chap. III, pg. 28, 1941.
3. Jordan, E. O.: General Bacteriology, W. B. Saunders Company, 12th Edition, pg. 52, 1938.
4. Case Reports of Massachusetts General Hospital, New England J. Med., 233:257, 1945.

SOME RECENT EXPERIENCES WITH AMEBIASIS IN CHILDREN*

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NEW ORLEANS

Our interest in amebiasis was aroused by the frequency with which it has been encountered among children presenting bizarre and equivocal symptomatology. Seventy-three examples of infection with cysts of *Endomaeba histolytica* were recently identified during a period of less than four months in routine office practice. Recognition of such common occurrence among children "not quite well but not really sick" should make the practitioner more alert to the possibility that he is dealing with amebiasis, and cause him to be diligent in appropriate diagnostic and therapeutic procedures.

In this infection, as in many others common in the young, remote effects and reflections in other body systems are the rule; among these 73 patients, there were but seven instances of mild dysentery, and only a few more had diarrhea of any significant degree. Symptoms are generally far more suggestive of a low grade systemic infection than of isolated intestinal disease. Three brief protocols will serve to illustrate disturbances which have come to arouse our suspicions; cysts of *Endamoeba histolytica* were recovered from each of these children as a routine procedure, and therapy accomplished relief of the peculiar symptoms, roughly coinciding with disappearance of parasites from the feces.

CASE NO. 1

L. M. A., a white girl of 3 years, had been under our care from the time of birth, and aside from occasional mild respiratory infections had always been well. Together with mild constipation, the parents now noted that the child had a capricious appetite, and that her disposition had changed. She seemed unusually irritable, cross, and difficult to manage; she did not enjoy the company of others, and had frequent temper tantrums. Examination showed little other than a peculiar complexion reminiscent of fading suntan, slight enlargement and tenderness of the liver.

CASE NO. 2

M. L., a white boy of 18 months, was brought to the office because of recent unexplained poor appetite and restless sleep. He was wilful and highstrung, with seemingly inexhaustible nervous energy, appeared to delight in defiant misbehavior and "baiting" of adults. Particularly distressing to the parents was the child's unaccustomed poor appetite and behavior at mealtime; he took particular delight in refusing morsels he had begged for only a short time before. He indicated the presence of transitory but apparently real abdominal pains. These were often severe enough to awaken him from a sound sleep or disturb play activities he otherwise enjoyed. These pains could not be localized by the child or his parents. While his usual mild constipation did not seem to be affected appreciably by laxative foods or simple medications, close questioning indicated that he had occasional loose mucoid stools, at times even blood-streaked. Complete examination revealed only a peculiar muddy complexion of the skin, moderate enlargement and tenderness of the liver;

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the sclerae and mucosae appeared entirely normal.

CASE NO. 3

M. G., a white boy of 3 years suddenly developed mild convulsive seizures which did not fit any recognized pattern. There had been no epilepsy or degenerative disease in the family. Appetite had been poor recently, and the child had had occasional diarrheic stools interrupting a commoner tendency to slight constipation. From his usual sunny, cheerful, outgoing behavior, he had become a sullen and retiring child. Physical examination demonstrated only a peculiar sallow complexion, together with a moderately enlarged and tender liver.

Aside from demonstrating cysts of *Endamoeba histolytica* in the fecal examinations, routine laboratory studies failed to reveal significant abnormalities; urinalyses and hemograms showed nothing unusual, serologic tests for syphilis were negative, and no abnormalities in the cephalin-cholesterol flocculation reaction were encountered.

In only one of these children, bowel disturbances had seemed noteworthy to the parents. All, however, had changes in bowel habits, behavior and customary disposition. Abdominal pain dominated the clinical picture in one child, convulsive seizures in the other. In contrast to the varying complaints for which these children were brought to us, the meagre physical findings were essentially similar. Changes after specific therapy for amebic parasites were remarkable.

The general features common to all 73 of these children included changes in appetite, established bowel habits, and personality. Many exhibited disturbed restless sleep, some had occasional diarrheic or loose stools, and in only a few (no more than three) was mucus or blood noted grossly. Nausea, vomiting, low grade fever, headaches insatiable appetite, increased fatiguability, weakness, and frequent colds appeared commonly in the listed complaints of parents. Abnormal pigmentation of the skin and equivocal tenderness of a slightly enlarged liver were frequently noted. All of these children were white; 35 were girls and 38 were boys. Three patients were encountered before the first birthday, nine were in their second year, 36 were between two and six years, and the other 25 ranged in age from six to 12 years.

Justification for this brief review of complaints so ordinarily considered protean lies in the fact that they existed together in patterns so variable as to defy orderly correlation. Only three conclusions seem warranted at present:

1. Alterations in accustomed personality, appetite, and bowel habits were invariably present.

2. A peculiar, though seemingly characteristic "fading suntan" complexion together with a slightly enlarged and tender liver constituted the only distinctive physical signs common enough to be noteworthy.

3. Complaints and abnormal physical signs subsided promptly with appropriate therapy.

We might add that there is need for clarifying further the cause and effect relationships of this disease in children, and that it deserves serious consideration when the diagnostician is faced with a puzzling hodge-podge of complaints that don't seem to fit together.

DISCUSSION

Dr. D'Antoni: Enlargement of the liver is an important manifestation of amebiasis in children; its rapid subsidence following therapy with non-absorbed amebicidal agents suggests that it is relatively rarely caused by intrahepatic growth of the parasites, but rather represents a transient toxic hepatitis. In adults, on the other hand, such hepatomegaly together with cysts of *endamoeba histolytica* in the stools would warrant a diagnosis of true amebic hepatitis. I have not any satisfactory explanation for the peculiar discoloration of the skin noted in many of these children, and have been particularly impressed with striking changes in personality and behavior coinciding with institution of therapy.

Not more than 20 per cent of all cases can be identified by examining normally passed stools; material secured after purgation and by sigmoidoscopy is far more satisfactory. We use a well buffered and highly effective saline purgative, and repeat sigmoidoscopy if the first examination is not conclusive.

In practically every family checked to discover the source of infection for these children, at least one adult in the household was found to harbor the parasite. Our studies suggest that it requires about six months of intimate contact for infection to become established in a new individual.

With *any* form of therapy, cures by our usual

laboratory criteria are notoriously difficult to attain in young subjects such as these. Because of their low toxicity, I have long favored the use of non-absorbed iodine preparations for children. We now give Diodoquin, which contains 63.9 per cent iodine, in full adult dosage to all children who weigh more than thirty pounds; we have no fear of toxicity with this schedule, comprising three tablets (3.2 grains each) three times daily for twenty days.

Dr. Webb: I think the authors have rendered a valuable service in pointing out the frequency of amebiasis in children and emphasizing its bizarre symptomatology, in which diarrhea is so often lacking. I do not believe we have encountered any proved examples of amebic abscess, which must be rare in young patients. Does ulceration of the intestinal tract occur very often?

Dr. D'Antoni: We do not expect it unless there is diarrhea; with rectosigmoidal amebiasis diarrhea usually occurs, and in a few such cases we have seen mild ulcerative lesions. If suspicion of amebiasis awaits the appearance of diarrhea, about 75 per cent of the cases will be missed.

Dr. Sanders: When do you call your patient well?

Dr. D'Antoni: When there is no longer an infection in the intestinal tract; though clinical improvement begins rather promptly after therapy is begun, the patient is not considered to be cured until the diagnostic routine I described has yielded negative results three times over a period of six months.

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A FAMILY OUTBREAK OF LEAD POISONING FROM STORAGE BATTERY CASINGS

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NEW ORLEANS

Lead poisoning has been recognized for years as an important pediatric and public health problem. Laws prohibiting the use of lead paint on toys and cribs have been followed by a sharp decline in prevalence. Other sources of lead intoxication in children still exist, however, and the following cases illustrate one of these.

The household consisted of a colored family of six who lived in the country in a one room shack with a stove and an open fire-place in the room. Water was obtained from a deep well. The father, aged 30, the mother, 24 years old, and four children, twins, a boy and a girl three years old, a girl of two years and a boy of one year, all were in good health before this episode. The family income was limited.

The children were breast fed for eight to nine months and had fairly good diets. Growth and development were normal and there was no history of pica. Previous illnesses had been limited to occasional, sometimes bloody, bouts of diarrhea and occasional brief febrile illnesses. No insect or rat poison had been used in the house and the parents were sure the children had had no other access to poison. The family dog went mad four weeks before the children became ill but had not bitten any of the children. The child that died and the one year old child had not played with the dog, but the others had. The father shot the dog but the head was not examined.

CASE NO 1

J. W., the 23 months old female, was brought to a private physician in Baton Rouge, Louisiana, on December 12, 1946, because of convulsions. The child was first noted to be ill three days previously, the chief symptoms being coryza and diarrhea. On December 11, the day before she was taken to the physician, she seemed to be very nervous, lethargic, weak, could not hold objects in her hands, and had several convulsions. At the time the physician examined the child, she had a temperature of 105.4°. Respirations were 65 and she was having severe convulsions. The patient was immediately referred to a local hospital but died on admission there. No information was available on physical or laboratory findings at that hospital.

CASES NO. 2 AND 3

Bobby W. and Betty J. W., the 3 year old twins, had almost identical stories. They were brought to Charity Hospital at New Orleans on December 13, 1946, the day after the death of the younger sister, chiefly because of this fact. The complaints at this time were limited to drowsiness, anorexia, irritability, and mild diarrhea, all of two days' duration.

On admission neither child had fever, the temperatures being 98.0° and 98.4°. Physical examination of both children revealed no abnormalities, other than the drowsiness and irritability. There was no papilledema, stiffness of the neck or other signs of disorder of the nervous system.

Because of the familial character of the outbreak and the history of diarrhea, the tentative

†From the Department of Pediatrics, Louisiana State University School of Medicine and the Pediatric Service of the Charity Hospital of Louisiana at New Orleans.

TABLE 1

LABORATORY DATA

	Betty J. W. 3 years	Bobby W. 3 years	Smiley W. 11 months
BLOOD			
RBC	3,350,000	4,900,000	3,830,000
Hgb.	9.0 gms.	11.2 gms.	10 gms.
WBC	10,500	12,900	5,850
PMN	39	49	55
L	57	45	38
M	1	0	3
E	3	6	4
SMEAR	Basophilic stippling present, 3 to 4 per high power field.		Basophilic stippling present, but rare.
URINE	Completely negative except for one plus reduction of Benedict's, which disappeared in all three cases on second examination.		
SEROLOGIC TEST FOR SYPHILIS	Negative	Negative	Negative
BLOOD SUGAR	89	78	....
SPINAL FLUID	....	Clear Pressure 100 mg. No cells Protein 50 mg. Sugar 133 mg. Chloride 708 mg.	....
X-RAY OF LONG BONES	All three cases showed marked linear increase in density in metaphysis adjacent to epiphyseal line.		

diagnosis at admission was possible shigellosis.

The laboratory data in these two children and the younger child are summarized in table 1.

With the discovery of stippled red blood cells the diagnosis of lead poisoning came to the fore. Further confirmation was obtained from the x-ray findings.

Because of the absence of serious symptoms in the children the need for immediate treatment as soon as lead poisoning was suspected was not appreciated. Three days after admission, however, Betty J. W. had a generalized convulsion lasting five minutes and followed by cyanosis. A second convulsion of the same duration appeared an hour later. There were no further convulsions subsequently. Phenobarbital, 30 mgms., was given for sedation.

Treatment directed at immobilizing the lead was begun promptly after the convulsion. The children were given a high calcium diet with a high phosphorus to calcium ratio. This included a quart of milk a day. Vitamin D was given in the form of calciferol, 5000 units daily. The children also received calcium lactate 0.65 grams t.i.d. and sodium citrate 2 grams t.i.d.

The other child never had any convulsions. The drowsiness and irritability improved steadily in both children and were completely absent 12 days after admission. They were discharged, apparently in good health, on December 28 after a total hospital stay of 15 days.

CASE NO 4

Smiley W., the 11 month old male, actually was the first in the family to become ill. The onset had been on December 8 with fever, irritability, vomiting, and diarrhea. He had ten to twelve watery bowel movements per day which contained quantities of mucus but no blood. Fever and diarrhea persisted until December 12, when he was hospitalized in Baton Rouge, Louisiana, chiefly because his sister had died that day. The patient had a convulsion the following day, but vomiting and diarrhea ceased in the hospital. He continued to be very irritable, however, and required sedation. A spinal tap was negative except for a slight elevation of pressure. No further laboratory data were available.

He was referred to Charity Hospital at New Orleans on December 18, after a diagnosis of lead poisoning had been made for the other children. The only positive findings at this time were marked irritability and pallor. There was no papilledema.

His treatment was essentially the same as that for his older siblings but sedation had to be carried out for a longer period of time because of his

marked irritability. No convulsions appeared in this hospital and his improvement was steady. He, too, was discharged on December 28, apparently in good health, after a total hospital stay of ten days.

COMMENT

The first clue to correct diagnosis in these cases was the finding of generalized stippling of the red blood cells. Credit for making this finding goes to a third year student who reported it promptly to the house physician. With the probability that the children were suffering from lead poisoning in mind, the parents were queried further regarding possible sources of the metal. No painting had been done in the house, there was no use of any pipe for drinking water, the children had no lead toys, and they did not chew on the furniture. Specific inquiry was then made regarding the fuel used in the fireplace and stove and it came to light that storage battery casings were being used almost exclusively. The father stated that they had used casings occasionally during the past three years when they could not obtain wood, but not for more than a day or two at a time. However, this winter they were unable to obtain wood and casings alone were burned for three consecutive weeks. The casings were burned in a wood stove in the room in which the children slept. Unfortunately no smears were made of the parents' blood but they had no symptoms of lead intoxication.

The first report of an epidemic of lead poisoning from the burning of battery casings was made by Williams, Schulz, Rothchild, Brown, and Smith[1] in 1933. They described 40 cases which occurred in poor homes in the city of Baltimore within a period of two months. Investigation disclosed that intermittent lead poisoning had begun to occur in the winter of 1930-31, when, with the economic depression reaching new low levels, junk dealers allowed poor persons to secure, free or at small cost, the casings of discarded storage batteries for fuel. These casings, which carried a considerable deposit of lead salts, burned freely and were an excellent source of heat, despite the penetrating and unpleasant odor produced. It is interesting to note that the original suggestion that storage battery casings might be at fault came from the uncle living in the house where the first child case was discovered. Cooperative public health educational efforts as a result of this epidemic apparently resulted in essentially eliminating the hazard in Baltimore. Several other cities took heed at the time and also carried on educational campaigns concerning this danger.

Other series of cases[2, 3] of lead poisoning from this source have been reported and it has been emphasized by Bourne,[4] particularly, that increased prevalence among negroes was probably due to their generally lower economic status.

A point of interest is the high proportion of children among all the series reported. At least two explanations are offered—one that children are in the house more than adults and therefore have quantitatively greater exposure, and the other, that there is proportionally greater absorption in children of inhaled lead fumes. McKhann[5] cites animal experimentation to support the thesis that the young of a species are more susceptible to lead intoxication than adults. Whatever the explanation, the greater incidence in children justifies extra precautions and continued educational efforts to prevent a serious and often fatal disease.

SUMMARY

1. The source of four cases of lead poisoning in one family was found to be the use of storage battery casings as fuel.

2. One patient died and the other three children had evidence of lead encephalopathy manifested by convulsions in two and irritability and drowsiness in one.

REFERENCES

1. Williams, H., Schulz, W. H., Rothchild, H. B., Brown, A. S., and Smith, F. R., Jr.: Lead poisoning from the burning of battery casings, J. A. M. A., 100:1485, 1933.

2. Alexander, A. J., and Downing, R. E.: Lead poisoning in children, report of an epidemic due to the burning of storage battery casings as fuel, Internat. Clinics, 2:229, 1941.

3. McDonald, J. M., and Kaplan, E.: Incidence of lead poisoning in city of Baltimore, J. A. M. A., 119:870, 1942.

4. Bourne, I. B.: Epidemic lead poisoning in children from storage casings, J. Nat. M. A., 36:187, 1944.

5. McKhann, C. F., and Vogt, E. C.: Lead poisoning in children, J. A. M. A., 101:1131, 1933.

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PEDIATRIC PROGRESS IN LOUISIANA

Prominent among developments in this specialty during the past twelve months was the formal reactivation of the Louisiana State Pediatric Society. Members have contributed generously to the annual program of the State Medical Society, as attested by a number of articles included in this issue. The group has met on two other occasions and has recently decided to hold regular meetings quarterly hereafter. An excellent program has already been planned for the next annual session at Monroe.

Dr. Sidney S. Chipman has recently come to Louisiana to serve as a member of the Pediatric staff at Louisiana State University Medical School and as pediatric consultant for the State Health Department. Both Tulane and L. S. U. Medical Schools are conducting many postgraduate activities, and at present have twenty-one graduate physicians enrolled as residents at Charity Hospital or as departmental fellows in training for completion of specialty Board requirements.

Through the cooperation of the State Health Department, the United States Children's Bureau, and the Board of Administrators of Charity Hospital, funds have been secured to permit expansion of facilities for care of premature infants. New construction, additional personnel and equipment bid fair to make this revised unit at Charity Hospital in New Orleans one of the largest and finest of its kind anywhere. An integral part of the program here is a plan for establishing a training center for graduate physicians and nurses. It is anticipated that teaching and research activities will lead directly to important advances in methods of caring for premature infants.

A new pediatric division at Hotel Dieu was formally dedicated on March 18, 1947, incorporating complete facilities for the care of 43 infants and children. On October 15, a pediatric ward was added at Foundation Hospital. A new pediatric wing is included in the Baton Rouge General Hospital now under construction; in the same city there are increased facilities for the care of children in the new Lady of the Lake Sanitarium. Appropriations have been made for establishing a special school for educable victims of spastic paralysis at Alexandria; this facility is now ready to receive its first pupils.

Collection of data for the Study of Child Health Services, sponsored by the American Academy of Pediatrics, is practically complete for Louisiana. We all owe a debt of gratitude to Dr. Clarence Webb, State Chairman for this study, to Dr. Verre Simpson, who served as Executive Secretary, and to numerous organizations and indi-

viduals interested in child welfare, without whose vigorous support this difficult project could not have been completed. Analysis of all schedules is now in progress, and Dr. Webb has appointed an Action Committee to interpret results and make specific critical recommendations. Their reports should appear in this Journal during the next few months.

Space does not permit a complete review of progress in pediatrics here; the foregoing brief notes serve only to point out a few praiseworthy activities which illustrate prevailing progressive attitude of workers in this field. Accomplishments within such a brief period augur well for the future of our state's richest natural resources—its infants and children.

NEONATAL DEATHS

Careful pathologic study of causes for death in prematurely born infants is the keystone on which effective clinical programs to reduce unnecessarily high mortality rates may be based. Conscientious investigators now have serious qualms about accepting prematurity alone as an adequate explanation for death. Continued improvement in survival rates for these small infants attests the fallacy of many empirically drawn criteria used to differentiate the premature from the mature infant or the previable from the viable fetus. There is general agreement that no foolproof criteria exist for such differentiations. Still, in all parts of the country, "prematurity" alone often appears— and is ordinarily accepted—as an adequate cause of death. Certainly, incomplete development is an important contributory factor, but in the majority, specific lesions can be disclosed. Failure to discover adequate anatomic bases for death in about 10 per cent of *mature* newborns tempts no one to inscribe "mature infant" as a cause on death certificates. Continuance of the practice of considering prematurity as more than a contributory factor calls to mind the classical head-in-sand performance of the ostrich.

Were it possible to carry out complete studies of all deaths among premature infants in the meticulous manner reported by Dr. Arey in this issue, there is no doubt that many fatalities now listed under "prematurity" or "cause undiagnosed" would be properly classified. Such studies as his should serve as a real stimulus for improving all aspects of our current programs directed at reduction of mortality among premature infants. We are in agreement with Potter, that at least 40 per cent of neonatal deaths are preventable on the basis of present knowledge—further knowledge and improved facilities can be expected to increase this percentage. This in turn depends on *more interest* and *more support* of more people in *more healthy infants*. Prevailing high death rates among our smallest citizens are not necessarily inevitable.

BEHAVIOR DISORDERS AND PHYSICAL DISEASE

Conduct disturbances are among the most common problems encountered by physicians who care for children. Their occurrence in the presence of physical disease is well recognized and the interesting report by Loeber and D'Antoni in this issue provides another apt illustration of this association.

It is important to bear in mind, however, that the genesis of such behavior disorders, mild or severe, is not easily explained. Those who are interested in phychosomatic medicine have repeatedly noted that psychic disorders both in children and adults disappeared after cure of physical disease, and, vice versa, that elimination of underlying psychic disturbances often resulted in disappearance of symptoms suggesting physical disease. It should not be inferred, therefore, that the occurrence of behavior disorders in children with amebiasis necessarily implies a specific causal relation. Parents who have been worried about a child's behavior, and whose worries in turn accentuate the behavior disturbances, may derive more psychologic benefit from being told that a definite diagnosis of physical disease has been made than from spe-

cific drug therapy given to the child. In an area of the country where parasitic infections are common and often quite symptomless, it is extremely difficult to relate minor changes in symptoms to presence or elimination of particular parasites. The need for controlled studies to elucidate this relationship further is emphasized by the high prevalence of infection with cysts of *Endamoeba histolytica* in children, shown by these authors.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

ANNUAL REPORT OF COMMITTEE ON RURAL HEALTH

Our Rural Health Committee is now in its third year and has been very active since its formation, three members of which attended meetings in Chicago.

Several meetings have been held with members of Rural Health Committee of the Farm Bureau in Louisiana and there has been organized an advisory council to the Farm Bureau, which council is composed of the following organizations.

Louisiana State Medical Society.
Louisiana State Medical Auxiliary.
Louisiana State Dental Society.
Louisiana State Department of Public Welfare.
Louisiana State Department of Education.
Louisiana State Board of Health.
Louisiana Parent-Teachers Association.
Louisiana Federation of Women's Clubs.
Louisiana Tuberculosis Association.
Louisiana Cancer Society (Division of American Cancer Society).
Louisiana Society for Crippled Children.
Louisiana Farm Home Administration.
Louisiana Agricultural Extension Service.
Blue Cross Association.
Louisiana Physician's Service.
Louisiana State Nurses Association.
Louisiana Home Demonstration Council.
Louisiana Hospital Association.
Louisiana Press Association.
Louisiana Department of Institutions.
Louisiana Experiment Station.
Associated Women of Louisiana Farm Bureau.
Louisiana Conference of Social Welfare.
Louisiana Farm Bureau.

Other statewide organizations or agencies which have a direct interest in rural health may be elected to membership by a vote of a majority of the members of the council.

Members at large may be elected by a majority vote of the members of the council.

A meeting was held in Baton Rouge June 22. Constitution and by-laws were drafted, in which the purpose is stated as follows:

"It shall be the purpose of the council to discuss problems relating to the health of the rural people of Louisiana, to prepare recommendations for the betterment of rural health as can be agreed upon by a majority of the members present at regular or special meetings, to advise and make recommendations to the Board of Directors of the Louisiana Farm Bureau with regard to their policies and programs for the betterment of rural health, and to sponsor the organization of local health councils."

At the State Medical Society meeting Mrs. T. R. Tomlinson, Chairman of Rural Health of Farm Bureau, was present and addressed the House of Delegates.

On Sunday, July 13 the American Academy of General Practice of Louisiana was organized. Dr. J. P. Sanders, a member of the Louisiana Rural Health Committee was elected president. Dr. Guy Jones was elected president-elect, Dr. D. B. Barber,

vice president and Dr. M. C. Wiginton a member of the Board of Directors.

The Louisiana State Medical Society has organized the Blue Shield Insurance Plan which is working with Blue Cross, and is now being offered to the rural areas.

A survey has been made in Louisiana of the hospital facilities of the state in conformity with requirements of the Hill-Burton Bill.

Assurance has been obtained that the medical schools in Louisiana will advocate a course in general practice and our hospitals will offer residencies in general practice which we hope will promote more and better general practitioners for rural areas.

On October 23 and 24 there was held in New Orleans the Southern Regional Conference of the American Medical Association Council on Medical Service. The Committee on Rural Health attended and on the night of the 23rd a banquet was held for found table discussion on Rural Health. This was well attended.

Respectfully submitted,

Guy R. Jones, M. D., A. A. G. P.

Chairman, Committee on Rural Health

REGIONAL CONFERENCE OPPOSES PROPOSED CONTROL OF MEDICINE BY GOVERNMENT

Keynoting the Southern Regional Conference meeting held in New Orleans recently was the unanimous opposition of the medical profession to a federal program of socialized medicine. The two-day Southern Regional Conference was sponsored by the Council on Medical Service of the American Medical Association in cooperation with the Council on Medical Service and Public Relations of the Louisiana State Medical Society.

"We are bitterly opposed to the socialization of medicine because in every country where the plan has been instituted it has brought an inferior grade of medical care," Dr. George F. Lull, Secretary and General Manager of the AMA and former deputy surgeon general of the U. S. Army, informed the conference.

Dr. Lull said that through the institution of prepaid medical and surgical care plans in the country any government attempt at socialized medicine can be staved off.

Dr. James McVay, conference chairman and chairman of the AMA's Council on Medical Service, explained the purpose of the conference and stated, "The AMA does not fight proposals for socialized medicine, we merely promulgate other methods of medical care which have proven superior.

"In voluntary prepayment plans such as the Louisiana Physicians Service, patients may choose their own doctor. A patient-physician relationship is firmly established. Under a federal program the patient would lose his identity. He would go to any doctor at a bureau. The patient would lose his individuality.

"One of the primary purposes of the conference is to plan for prepaid medical care for everyone in the country through a method of budgeting medical expenses."

Both Drs. Lull and McVay praised the Hill-Burton program, which has made extensive surveys into hospital and health facilities in U. S. rural and urban areas.

Dr. McVay came to New Orleans from Kansas City and Dr. Lull from AMA headquarters in Chicago.

The conference fostered the interchange of ideas on Public Relations, Prepayment Medical and Surgical Care Plans, Industrial Health and Rural Health.

The portion of the program devoted to Public Relations was headed by Henry Johnson, Public Relations Director for the Medical Society of Virginia. Five panel discussants actively participated in the public relations program and presented papers and answered questions expounded by members attending the conference.

Dr. Charles R. Henry of Little Rock, Arkansas, was moderator for the portion of the conference devoted to the discussion of prepayment medical and surgical care plans. More than 65 plans are now sponsored by either a state or county (parish) medical society, providing a medium where-

by more than seven million persons may prepay the cost of their anticipated medical bills.

Inauguration of a medical school program on prevention and treatment of diseases related to atomic energy was urged by Dr. Jean Felton, health department superintendent at the University of Chicago laboratory at Oak Ridge, Tennessee, atomic bomb plant. Dr. Felton participated in the Industrial Health portion of the conference. Dr. Raymond Hussey of Detroit, vice-chairman of the AMA Industrial Health Council, was moderator at the Industrial Health session.

The general practitioner was described as "the work horse of American Medicine" by Dr. J. Paul Jones of Camden, Alabama, member of the AMA's Rural Medical Service Committee and moderator for the Rural Health portion of the conference program.

Dr. Jones stressed the following as "pressing" rural health problems.

1. Hospital facilities and health centers.
2. Voluntary prepayment plans.
3. Nursing needs of rural communities.
4. Methods of bringing and holding doctors and dentists in rural areas.
5. Health councils as an agency for promoting rural health.
6. Medical care for low income groups.

A rural health panel discussant, Dr. William C. Chaney of Memphis, Tennessee, emphasized the fact that "People would like to see the physician a little more human; human enough to come into their homes when they are seriously ill. After all, people can die at home," he asserted.

Turning to what he labeled as a serious problem facing the medical profession today, Dr. Chaney said:

"The startling fact that our national government in 1946 spent $75,000,000 of the taxpayers' money to spread propaganda favoring the government control of the practice of medicine should bring every physician, dentist, pharmacist and nurse to his feet with the shocking realization that American medicine . . . may soon come to an end unless we do something about it."

The conference was attended by delegates from Louisiana, Mississippi, Alabama, Arkansas, Oklahoma, Tennessee and Texas.

The Council on Medical Service and Public Relations of the State Society handled local arrangements for the conference. Members of the Council are: Dr. A. .V Friedrichs, Chairman; Dr. P. T. Talbot, Dr. Daniel J. Murphy, all of New Orleans; Dr. John G. Snelling of Monroe; Dr. O. B. Owens of Alexandria and Frank Lais, Jr., Executive Director, New Orleans.

—o—

STATE PREPAYMENT PLAN CELEBRATES FIRST ANNIVERSARY

Louisiana Physicians Service celebrated its first anniversary on November 1. The first prepayment surgical and obstetrical care plan contracts issued by the company became effective November 1, 1946.

During the past year the company has been able to enroll nearly 20,000 individuals. In the same period, 1,965 claims were serviced. In the servicing of these claims the plan has paid to doctors throughout Louisiana in excess of $95,000.00 in claims for services rendered to LPS subscribers and their dependents.

Greatest utilization has been on the following listed services: Tonsillectomy and adenoidectomy; obstetrics, appendectomy, hysterectomy, anesthesia and x-ray.

The plan spreads the costs of medical care over a long period of time and among individuals through the application of the insurance principle. Through this medium a person can budget his anticipated medical needs—his anticipated doctor bills by paying a small amount each month into a common fund on a prepayment and post-payment basis—just as he would purchase any tangible asset on a part-payment or installment basis.

The subscriber may choose his own doctor and the doctor remains free of bureaucratic restrictions on his methods of treatment. It is anticipated that plans of this type throughout the nation will be an ally and instrument in the physicians'

hands in preserving the practice of medicine as it is known today and will forever kill the threat of socialization of medicine.

Dr. H. Whitney Boggs, Secretary-Treasurer of the plan recently stated that "desires often are more expensive than needs. The doctor gets only 13 cents out of each dollar the public spends for health. The remainder goes to medicine manufacturers, leaders of health cults, druggists, hospitals and others."

Dr. Boggs pointed out that people spend more for cigarettes, cosmetics, liquor and other luxury items than for doctor bills. But because medical expenses come all at once and are not always expected, many cannot pay them. More than 41 per cent of the population is in an income group which does not permit them to meet major doctor bills.

The remaining problem remains for an all-out presentation of the plan to the general public throughout Louisiana. We request that each physician take a personal interest in the presentation of his plan and encourage his patients to inquire about its protection. Within a few weeks printed material will be available and we ask that as many physicians as possible contact our office for a supply to be included in next mailing of monthly statements to patients.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

December 8,	Orleans Parish Medical Society 8 p. m.
December 9,	Orleans Parish Radiological Society, 7:30 p. m.
December 10,	Woman's Auxiliary, Orleans Club, 3 p. m.
	Touro Infirmary Staff, 8 p. m.
December 11,	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
December 15,	Hotel Dieu Staff, 8 p. m.
December 16,	I.C.R.R. Hospital Staff, 12:30 p. m.
	Charity Hospital Medical Staff, 8 p. m.
December 17,	Charity Hospital Surgical Staff, 8 p. m.
December 18,	New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
	Veterans Administration Hospital Staff, 8 p. m.
December 19,	Lakeshore Hospital Staff, 8 p. m.
December 22,	DePaul Sanitarium Staff, 8 p. m.
December 23,	Baptist Hospital Staff, 8 p. m.
January 2,	Foundation Hospital Staff, 8 p. m.
January 5,	Board of Directors, Orleans Parish Medical Society, 8 p. m.
January 6,	Eye, Ear, Nose and Throat Staff, 8 p. m.
January 7,	Mercy Hospital Staff, 8 p. m.
January 8,	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
January 12,	Installation Meeting, Orleans Parish Medical Society, Jung Hotel, 7:30 p. m.

NEWS ITEMS

The following members of the Society participated in the program of the Southern Psychiatric Association in Birmingham, October 13-14:

Dr. John W. Bick, Jr., spoke on the "Adjustment Problems in the Modern Woman"; Dr. Hunter Harris read a paper prepared by Dr. H. O. Colomb on "The Transference Mechanism in Medical Practice"; and Dr. Theo. A. Watters spoke on the "The Ego and the Eye."

Dr. Walter Otis, who also attended this meeting, was installed as vice-president of the organization.

Dr. Theo. A. Watters recently attended the anual conference of the Menninger Foundation at Topeka, Kansas. Before returning to New Orleans, Dr. Watters visited the Winter Veterans Hospital, where the largest training program for psychiatrists in the world is going on—120 being trained at the present time.

Dr. Ruth M. Shushan has just returned from New York where she was a member of the first class in cytologic diagnosis of cancer, given by Dr. George Papanicolaou at Cornell University Medical College.

Dr. Edmond Souchon was recently elected a fellow in the International College of Surgeons.

Dr. Charles L. Eshleman was elected president of the medical staff of Foundation hospital on September 26. Other officers elected were: Dr. A. Seldon Mann, vice-president; Dr. Robert C. Lynch, secretary-treasurer; Drs. Thomas Findley,

L. S. Meriwether and I. Mims Gage, members of the executive committee.

Dr. E. J. Richard was re-elected as head of the independent unit staff at Charity hospital on September 24. Dr. Blaise Salatich was elected secretary. Heads of departments named were: Dr. F. F. Boyce, surgery; Dr. Adolph Jacobs, obstetrics; Dr. George Battalora, orthopedics; Dr. Monroe Wolf, urology, and Dr. J. Roeling-Hanley, orthorhinolaryngology. Three staff members named were: Drs. John F. Oakley, N. J. Tessitore and E. L. Zander.

The following officers of the Charity Hospital Visiting Staff were elected at the annual meeting of the Staff held October 2: Dr. P. H. Jones, Jr., president; Dr. J. D. Rives, vice-president; Dr. Nathan Polmer, secretary; Drs. John Oakley, Edgar Hull, Conrad Collins and E. J. Richard, members of the medical advisory committee.

At a recent meeting of the Metairie Foundation Hospital Dr. Joel B. Gray was elected president of the staff. Other officers elected are as follows: Dr. Louis J. Gehbauer, vice-president; and Dr. Wallace C. Beil, secretary-treasurer.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

RAPIDES PARISH MEDICAL SOCIETY

The Rapides Parish Medical Society is celebrating on January 10, 1948 their "Founders Day" by putting on a top-notch full day's program under the name of "Founders Day Forum". There are to be scientific papers beginning at 10:00 a. m., a luncheon at 12:15 p. m. and an afternoon session which will end at 4:30 p. m. Appearing on the program are some of the outstanding specialists and teachers on the staffs of Louisiana State University and Tulane medical faculties. In the evening the annual banquet will be held and the group will be addressed by an outstanding guest speaker. The Bentley Hotel, Alexandria, is the site for the all day program.

Certainly the doctors of Rapides Parish and the members of the arrangement committee are to be congratulated on this unique and outstanding program. All members of the profession should make plans now to attend and take advantage of this scientific, cultural and social opportunity—a real red letter day in medical progress.

SIXTH DISTRICT MEDICAL SOCIETY

Members of the Sixth District Medical Society held their semi-annual meeting at East Louisiana State Hospital in Jackson, November 7. Members present were guests of the Hospital at supper and then retired to a scientific program.

Dr. Charles E. Sturm of the Hospital staff presented a paper on shock therapy. Dr. Charles S. Holbrook of New Orleans spoke on statistics pertaining to shock therapy. Fractures of the elbow were discussed by Dr. I. L. George of Baton Rouge.

NATIONAL CONFERENCE OF COUNTY MEDICAL SOCIETY OFFICERS

The Grass Roots Conference of the National Conference of County Medical Society Officers, the purpose of which is to develop a working partnership between the American Medical Association and all physicians, will be held at the Statler Hotel in Cleveland, Ohio on January 6. Subjects to be discussed are: 1. The General Practitioner; How to Create More of Them for the Future Need of the Country; 2. Upholding the Prestige of the General Practitioner; 3. The General Practitioner and Community Leadership. Hotel reservations should be requested through Dr. David Chambers, Chairman Hotel Committee (A.M.A.), 511 Terminal Tower, Cleveland 13, Ohio.

SIXTH DISTRICT AMERICAN ACADEMY OF GENERAL PRACTITIONERS ORGANIZED

An organization meeting of the American Academy of General Practice in the Sixth District

was held November 7, at the East Louisiana State Hospital in Jackson. The meeting was held immediately preceding the semi-annual meeting of the Sixth District Medical Society.

Officers elected by the group are as follows: Dr. M. C. Wiginton of Hammond, President; Dr. A. L. Lewis of Amite, Vice-President; Dr. Charles J. Wise of Angola, Secretary-Treasurer. Arrangements for the election of eight directors to the board of the Sixth District A.A.G.P. were completed and the board is expected to function as such by the time of the next meeting. It is anticipated that each organized medical society in the district will have one representative on the board.

Dr. D. B. Barber of Alexandria, Vice-President of the State A.A.G.P. talked briefly on the benefits of the general practitioner becoming a member of his district organization. Dr. Joel Gray of New Orleans spoke on the Constitution and By-Laws. Dr. O. W. Topp, recently elected President of A.A.G.P. of the Seventh District, talked on the organization of that district.

Dr. Wiginton reported approximately thirty members enrolled from the Sixth District.

PUBLIC RELATIONS PROGRAM PLANNED BY SHREVEPORT SOCIETY

Reports are that the Shreveport Medical Society through its Public Relations Committee expects to establish an active Public Relations program in the Shreveport area right after the first of the year.

A luncheon meeting was recently held in Shreveport to formulate tentative plans for the establishment of the program in cooperation with the proposed Public Relations program of the State Society. Shreveporters interested in Public Relations attending the meeting were: Dr. Jos. E. Heard, President of the local society, Dr. R. Denman Crow, Dr. W. G. Wells, Dr. A. A. Herold, Dr. H. Whitney Boggs and two members of the Public Relations Committee, Dr. J. R. Stamper and Dr. J. P. Sanders. Mr. Frank Lais, Jr., Executive Director of the Council on Medical Service and Public Relations, also attended and represented the Council.

Mr. Lais advised the group that numerous prepared recordings are available from AMA headquarters in addition to numerous other mediums of promoting public relations. He suggested the establishment of speaker bureaus in order that the doctor may present to the public his opposition to the socialization of medicine. Dr. Boggs advised the group that it was his belief that the Shreveport Chamber of Commerce would assist in the presentation of their program.

PREPAYMENT PLAN EXPLAINED TO SHREVEPORT AUXILIARY

An open forum, sponsored by the Woman's Auxiliary of the Shreveport Medical Society, was held recently in the State Exhibit Building in Shreveport. Features, benefits and economics of a prepaid medical insurance plan were explained by three officers of Louisiana Physicians Service: Dr. H. Whitney Boggs, Secretary-Treasurer, Dr. O. B. Owens, President, and Frank Lais, Jr., Executive Director.

Dr. Boggs said that medical economics involves persons in the low-income brackets, who have to struggle to satisfy their economic wants. Economics, he stressed, is a study of a person's needs and/or his desires. By spreading financial risk, the prepaid medical plan makes it possible for persons to meet their medical obligations.

Pointing out that Louisiana Physicians Service spreads medical costs over a long period of time and among individuals, the speakers declared that it provides low-cost service and that it is the answer to threats of socialized medicine.

Dr. Owens asserted that over a long period of time, the medical profession has been threatened with the possibility of having forced upon it a federal bureaucratic control, or dictatorial system, of the distribution of medical care. Heeding this menace, the doctors of the nation have organized their own plans, advocating free enterprise.

NEWS ITEMS

Dr. Robert A. Katz has returned from England and the Continent, where he has been for the past two months. While there, he reported on his researches on arteriosclerosis.

On November 2 and 3, he presented in Chicago, to the American Society for the Study of Arteriosclerosis, two addresses: "The Status of Arteriosclerosis in Europe" and "Intravenous Ether, a New Approach to the Therapy of Arteriosclerosis."

INTERNATIONAL COLLEGE OF SURGEONS

At the Twelfth Assembly and Convocation of the United States Chapter, International College of Surgeons, held in Chicago on October 3, the following Louisiana doctors were among the 810 surgeons inducted into the College:

FELLOWS: Peachy Ridgway Gilmer, M. D., Shreveport; John A. Hendrick, M. D., Shreveport; Claude A. LaRue, M. D., Shreveport; Frank C. Shute, Jr., M. D., Opelousas; Edmond Souchon, II, M. D., New Orleans; John Wm. Faulk, Sr., M. D., Crowley; Wilton Paul Duncan Tilly, Sr., M. D., New Iberia.

ASSOCIATES: Charles V. Hatchette, M. D., Lake Charles; William V. Garnier, M. D., Bastrop; Charles McVea, M. D., Baton Rouge; William Henry Roeling, M. D., New Orleans; William McGinty McBride, M. D., Alexandria.

AFFILIATES: Jay W. Cummins, M. D., Monroe; Joseph A. Vella, M. D., New Orleans.

MATRICULATE: Richard Denman Crow, M. D., Shreveport.

COLLEGE OF AMERICAN PATHOLOGISTS

The first general meeting of the College of American Pathologists was held in Chicago on October 27. A dinner meeting was held at the Drake Hotel after which there was formal introduction of members of the College at a meeting in the John B. Murphy Memorial Auditorium. Included were the following physicians from Louisiana: Dr. John L. Beven, Baton Rouge; Dr. Willis P. Butler, Shreveport; Drs. S. H. Colvin, Charles E. Dunlap, George H. Hauser, Russell L. Holman, Edwin H. Lawson, Aldea Maher, Emma S. Moss and Phillip Pizzolato; all of New Orleans.

The objects of this new organization are to (a) foster the highest standards in education, research, and the practice of pathology, (b) through study, education, and improvement of the economic aspects of the practice of pathology to advance the science of pathology and to improve medical laboratory service to physicians, to hospitals and to the public, (c) to maintain the dignity, precision and efficiency of the specialty of pathology for the service of the common good.

SCIENTIFIC EXHIBIT AWARD

Scientific exhibit, entitled "The Etiologic Agents of Mycotic Infections; Laboratory Methods of Identification and Classification" prepared by Drs. Albert L. McQuown and Emma S. Moss, of New Orleans, merited a silver medal at the 1947 meeting of The American Society of Clinical Pathologists held in Chicago October 27-30.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

The National Society for the Prevention of Blindness will hold its 33rd Annual Meeting on December 12, in the Russell Sage Foundation Building, 130 East 22nd Street, New York City.

The principal speaker will be Dr. C. E. A. Winslow, Editor of the *American Journal of Public Health*. Dr. Winslow's subject will be "Prevention of Blindness in the Public Health Program."

Activities of the National Society for the Prevention of Blindness during the past year will be reviewed by Dr. Franklin M. Foote, Executive Director and Mason H. Bigelow, President of the Society, will preside.

EIGHTH ANNUAL CONGRESS ON INDUSTRIAL HEALTH

The Council on Industrial Health will hold its Eighth Annual Congress on Industrial Health in the Cleveland Auditorium, Cleveland, on January 5-6, 1948. These dates immediately precede the Interim Session of the American Medical Association, which will be held in the Auditorium on January 7-8. General practitioners supply a large part of the medical services which workers receive through industry, and they are cordially invited to attend these industrial health sessions. The program of the Congress is being constructed with general practitioners in mind and will include discussions of first aid and emergency services in industry, physical examinations, administrative practices, applied physiology, aviation medicine, radiation medicine and practical expositions of occupational disease management, traumatic surgery and rehabilitation. Since full use of medical services in industry depends on support from management and the worker, the essential relationships will be discussed. Industry needs medicine as a practical ally and to promote human relations. The Industrial Health Congresses are intended to further these objectives.

EYE SPECIALISTS OF THE AMERICAS TO MEET IN HAVANA

Plans have been announced for the Third Pan-American Congress of Ophthalmology, which is to be held in Havana, Cuba, January 4-10, 1948. It is anticipated that about 1,000 eye specialists will attend the sessions at the University of Havana School of Medicine.

A program of more than forty formal papers has been arranged, with speakers divided about equally between physicians of the Northern half of the hemisphere and those of the Latin American countries. Papers presented by English-speaking ophthalmologists are to be discussed by their colleagues who speak Spanish, Portuguese or French, and the papers in the Latin languages will be discussed by members of the English-speaking contingent. During the presentation of each paper, slides bearing resumés of one other language will be shown, in order to facilitate understanding by those who are unfamiliar with the language used by the speaker.

Topics on which the eye physicians will exchange information cover a wide range. They include operations for cataract, glaucoma and crossed eyes, parasites that attack the eyes, hazards to the eyes in industry, effects of high blood pressure, syphilis and certain tropical diseases on the eyes, tumors, sulfonamide and penicillin treatment, and diseases peculiar to high altitudes of such countries as Peru and Bolivia.

MIDWINTER SEMINAR IN OTOLARYNGOLOGY AND OPHTHALMOLOGY

The University of Florida Midwinter Seminar in Otolaryngology and Ophthalmology will be held at the Flamingo Hotel in Miami Beach, beginning on January 12-17, 1948. Registration fee will be $25.00.

Distinguished lecturers for the courses in Otolaryngology include Drs. Lawrence R. Boies, Minneapolis; Louis H. Clerf, Philadelphia; Kenneth M. Day, Pittsburgh; Thomas C. Galloway, Chicago; James H. Maxwell, Ann Arbor; Arthur W. Proetz, St. Louis; and Harry P. Schenck, Philadelphia. Among the outstanding ophthalmologists who will lecture are Drs. S. Judd Beach, Portland, Me.; William L. Benedict, Rochester, Minn.; Daniel B. Kirby, New York; Peter C. Kronfeld, Chicago; and Donrmann K. Pischel, San Francisco.

The Midwinter Seminar follows immediately the Pan-American Congress of Ophthalmology, which will be held in Havana, Cuba, January 5-10, 1948. The dates chosen for the two meetings make possible a delightful opportunity to attend both and at the same time enjoy a winter vacation amid unsurpassed resort attractions.

MICHIGAN POSTGRADUATE CLINICAL INSTITUTE

The Second Annual Michigan Postgraduate Clinical Institute will he held at the Book-Cadillac Hotel, March 10-12, 1948. Forty-nine outstanding clinicians and lecturers will present a concentrated three-day postgraduate course covering the newest developments in medicine, surgery, obstetrics, pediatrics, dermatology, ophth-otolaryngology and general practice.

Two evening sessions will be held, the Wednesday night presentation being a "question box" and the Thursday evening program being a panel discussion on "First Aid to the Acutely Injured Patient".

All members of the American Medical Association and of the Canadian Medical Association are cordially invited to attend. There will be no registration fee.

SOUTHEASTERN SURGICAL CONGRESS

The Sixteenth Annual Assembly of The Southeastern Surgical Congress will be held in Hollywood, Florida, Hollywood Beach Hotel, April 5-8, 1948. Included in the list of men who will appear on the program are the following members of the Louisiana State Medical Society: Drs. F. F. Boyce, W. E. Kittredge and Ambrose Storck; all of New Orleans.

SOCIETY FOR THE PREVENTION OF ASPHYXIAL DEATH, INC.

For many years Dr. Chevalier Jackson has urged that all physicians become familiar with the "death zone of the respiratory tract." For the last fifteen years the Society for the Prevention of Asphyxial Death, Inc. has labored to make the physician "asphyxia conscious." Since April of this year, classes in laryngoscopy and intubation have been given at the Manhattan General Hospital in cooperation with the S.P.A.D., Inc. Other courses are planned for the New York Eye and Ear Infirmary.

Wide spread interest in these courses, approved by the Council on Medical Education and Hospitals of the American Medical Association, is suggested by the enrolment of physicians in the following specialties who have taken this course—Anesthesia, Eye, Ear, Nose and Throat, Internal Medicine, Industrial Medicine, Obstetrics, Gynecology, Pneumatology, Pediatrics, Plastic Surgery, Oral Surgery, Chest Surgery, General Surgery. These physicians came from Canada, Colorado, Connecticut, Florida, Iowa, Indiana, Illinois, Maryland, Massachusetts, Michigan, New York, New Jersey, Nebraska, Ohio, Pennsylvania and Virginia.

The course stresses the stages of asphyxia, the pathologic physiology of each stage and the indications for treatment. Resuscitation apparatus and resuscitation gadgets are considered important only in so far as they meet the pathologic indication for treatment.

The purpose of the course is clear cut. It is to save lives now lost during anesthesia and other forms of acute asphyxial accidents. Classes are limited to 12 students. A course will be given December 5-6 and January 2-3 in New York City; January 14-15 in Los Angeles; January 21-22 in Honolulu, Hawaii; February 6-7, March 5-6 and April 2-3 in New York City. For further information address: Pneumatology; Manhattan General Hospital, 307 2nd Avenue, New York City.

THE EYE-BANK FOR SIGHT RESTORATION, INC.

Fellowships for research in ophthalmology have been granted to the medical schools of Harvard and Yale Universities by The Eye-Bank for Sight Restoration, Inc., it has been announced by Mrs. Aida de Acosta Breckinridge, Executive Director.

Recipients of fellowships will devote themselves chiefly to problems related to the cornea. It is expected that the knowledge thus acquired will aid in the conservation of vision and the restoration of sight among thousands of individuals. One of the principal objectives of research carried on at the present time is the discovery of a method for the preservation of corneal tissue for a period longer than 72 hours.

The Eye-Bank for Sight Restoration, Inc., collects healthy corneal tissue from human eyes for transplanting to blind persons who have lost their sight because of corneal defects. Its national headquarters are located at 210 East 64th Street, New York City, where it maintains its own research laboratory.

The operation substituting a healthy cornea for a damaged one can restore sight in only one type of blindness—that caused solely by opacity of the

cornea when the rest of the eye and optic nerve are normal.

FELLOWSHIPS FOR POSTGRADUATE STUDY

The National Jewish Hospital at Denver announces a program of Fellowships for postgraduate study in tuberculosis and allied diseases. Fellows will be appointed for three-month, six-month or one year periods. Further information can be obtained by communicating with Dr. Edgar Mayer, Chairman, National Medical Advisory Board, National Jewish Hospital at Denver, 470 Park Avenue, New York or to Dr. Allan Hurst, Medical Director National Jewish Hospital at Denver, 3800 East Colfax Avenue, Denver.

INSTITUTUM DIVI THOMAS

Importance of clinical medicine in scientific research is emphasized by the Institutum Divi Thomas, Cincinnati, O., graduate research institution, in its recent appointment of a Clinical Advisory Committee.

Nine outstanding physicians and scientists from various parts of the United States have been appointed to the Clinical Advisory Committee, according to an announcement by Monsignor Cletus A. Miller and Dr. George S. Sperti, president and director respectively, of the Institutum Divi Thomas.

This committee will serve in a consultant capacity to the Institutum in the clinical aspects of research, carried on in laboratories of the Institutum and its 13 affiliated units.

The Institutum Divi Thomas, founded in 1935 by the Catholic Archbishop of Cincinnati and Dr. Sperti, is devoted to fundamental scientific research. Its scientific activities, at the graduate level, are carried on in the Institutum laboratories in Cincinnati and Palm Beach, and at 13 laboratories in various parts of the United States which are affiliated with the Institutum's research program.

There is an affiliated unit at St. Mary's Dominican College, New Orleans.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, from the figures secured from the New Orleans Health Department, reported that during the week which ended October 11, 137 persons died in the City of New Orleans. Of these 76 were white individuals, 61 nonwhite and eight were children less than one year of age. The next week, ending October 18, showed an increase in number of deaths, there being 179 reported this week. One hundred and fourteen of these deaths occurred in the white population and 65 in the colored. Twenty-six deaths occurred in infants this week, divided 12 and 14 between the white and colored races, respectively. There was a decrease of 47 in the number of deaths reported for the week which came to a close on October 25. Eighty-eight white persons died this week and 44 deaths were reported in the nonwhite population. Death of 11 white infants and six colored children was reported during this particular week. There was only one more death reported during the week which terminated November 1, over the number reported the preceding week. These were divided 81 white, 52 nonwhite and 12 infant deaths; divided equally between the two races.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending September 27 indicated that cancer and pulmonary tuberculosis were still the most prevalent reported diseases occurring in the state. There were 37 cases of cancer reported and 35 of pulmonary tuberculosis. These were the only two diseases reported in numbers greater than ten. Undulant fever occurred in two parishes; Lafourche and Washington Parishes each reported one case. There was one case of meningococcus meningitis reported in Jackson Parish and one case of poliomyelitis in East Baton Rouge. The following week, ending October 4, the report showed cancer still at the top of the list with 45 cases, followed by 28 cases of pulmonary tuberculosis, 16 of unclassified pneumonia and 11 of whooping cough. Two cases of malaria were reported and it was stated that both of these were contracted outside of Continental United States. There were no poliomyelitis cases reported this particular week, however one case of meningococcus meningitis was reported in Rapides Parish and two cases of undulant fever were reported in Washington Parish. Measles led all reported diseases on the list for the week ending October 11. There were 49 cases of this disease reported followed by 26 of cancer, 23 of pulmonary tuberculosis and 11 of unclassified pneumonia. Again there were no cases of poliomyelitis reported. One case of meningococcus meningitis was reported from Ascension Parish. There were 50 cases of pulmonary tuberculosis reported for the week which terminated October 18, followed in order of frequency by cancer with 20 cases, unclassified pneumonia with 16 and hookworm infestation with 11. One case of poliomyelitis occurred in Orleans Parish and no cases of meningococcus meningitis were reported this week. Cancer was again at the top of the list, with 47 cases reported the week ending October 25. Pulmonary tuberculosis closely followed with 44 cases and then unclassified pneumonia with 13; these being the only diseases listed in numbers greater than ten. Two cases of poliomyelitis were reported this week; one from Morehouse Parish and one from Orleans. The same order of frequency of cases of cancer, pulmonary tuberculosis and unclassified pneumonia

occurred during the week which ended November 1, these being as follows: cancer 52, pulmonary tuberculosis 50 and unclassified pneumonia 36. One case of meningococcus meningitis was reported from St. Bernard Parish and two cases of poliomyelitis, one each from East Baton Rouge and Livingston Parishes.

MONTHLY MORBIDITY FOR VENEREAL DISEASES STATE OF LOUISIANA

Month Ending September 30, 1947

	Total This Month	Total Previous Months	Total To Date 1947
CHANCROID	53	433	486
GONORRHEA	1101	10040	11141
GRANULOMA INGUINALE	11	141	152
LYMPHOPATHIA VENEREUM	9	62	71
SYPHILIS	776	7679	8455

HARRY JOHNSTON

1870-1947

Dr. Harry Johnston, of Baton Rouge, died on September 26. He had been an active member of the State Society and the East Baton Rouge Parish Society for many years and will be greatly missed by members of the medical profession. Dr. Johnston graduated from the Tulane University School of Medicine in 1900.

LIONEL O. WAGUESPACK

1878-1947

Dr. Lionel O. Waguespack, of Vacherie, an active and interested member of the Second District and State Medical Societies, died at his home on November 3, 1947. Dr. Waguespack practiced medicine in Louisiana for many years, having graduated from Tulane in 1900.

THOMAS E. WILLIAMS

1873-1947

Dr. Thomas E. Williams, a member of the Shreveport and State Societies since 1918, died on October 4. Dr. Williams graduated from the Jefferson Medical College in Philadelphia in 1905 and spent most of his years of medical practice in Louisiana.

COMMUNICATION

The Editor,
New Orleans Medical and Surgical Journal,
1430 Tulane Avenue,
New Orleans 13, La.
Dear Sir:

I have been requested by the Board of Administrators of Charity Hospital of Louisiana at New Orleans to clarify the remarks concerning the use of endotracheal anesthesia for teaching purposes in my recent article on "Thiouracil in Toxic Thyroid Disease: Its Preoperative Use in a Public Hospital in a Non-endemic Area," read before the Orleans Parish Medical Society April 14, 1947, and published in the New Orleans Medical and Surgical Journal for August, 1947.

No statement in this article is intended to imply that any method of anesthesia was used in any case in the reported series without the consent and approval of the operating surgeon, on the ground that it was for the interests of the patient that this type of anesthesia should be used. Nor should any statement in the article be construed to mean that the teaching of anesthesia ever superceded the interests of the patient, although the administration of endotracheal anesthesia was taught incidentally in many cases in the series.

These considerations, it seems to me, are self-evident. There is universal agreement that the expert medical care of patients and the education of physicians along special lines not only are not incompatible but actually complement each other. There is also universal agreement that patients cared for in teaching hospitals—which Charity Hospital is accredited as—and used for teaching purposes receive medical care of the highest quality because they are supervised with particular care.

Although, as I have said, these facts are to me so self-evident that they need no elaboration, questions have apparently arisen concerning the statement in my article dealing with the teaching of anesthesia in the New Orleans Charity Hospital. Certainly this statement was not intended to impugn the motives or the judgment of any surgeon who employed it (I happen to use it very frequently myself) nor was it intended in any way to reflect upon the Department of Anesthesia of the Hospital.

I shall appreciate the publication of this note.

Very truly yours,
Frederick F. Boyce, M. D.

WOMAN'S AUXILIARY, LOUISIANA STATE MEDICAL SOCIETY

The Louisiana State Medical Society has adopted as a postwar activity "Extension of Medical Services through the Louisiana Physicians Service, Inc." We, as members of the Woman's Auxiliary to the Louisiana State Medical Society, are vitally

interested in our own plan. Results are what we are interested in primarily; results not of today, but tomorrow and tomorrow's tomorrow. To achieve these results we must have sound planning and a thorough knowledge of our aims and policies. We must not expect overnight results in our public relations activities, but we must have patience and keep on working. By repetition facts will be stamped on the minds of the public.

First, we must educate ourselves. Begin this study in your own home by informing yourself; learn to know your subject matter so well you can discuss it with confidence and at ease on any occasion. Discussing current medical problems at home will serve two purposes. It will develop your ability to talk convincingly to the public, and it will contribute to your husband's knowledge of what is being done along these lines. Your next step is to coordinate your knowledge with that of other auxiliary members at your regular meetings.

There are many proposed bills before our National Congress today. Among them is the Truman Health Bill. The Murray-Wagner-Dingell Bill is expected back in a new frock. We know the aim of the medical profession is to put good medicine within the reach of all people. We should let others know of this undertaking. Any medical care plan must be organized on a scientific basis and operated under medical supervision. It is no longer a question of a change in medicine; the change is before us. It is now a question of who will shape the operation of the plan. We must not fail!

Many states are finding prepayment medical plans very successful. However, all these projects need publicity and the public must be adequately informed. The medical profession has demonstrated its ability to do a good job by the high quality of its service to mankind. The government should not be allowed to usurp the administration of medical care. The public is beginning to comprehend! It is alert, and as auxiliary members we must keep informed of what prepayment benefits are, and how they can be obtained through Louisiana Physicians Service, Inc. and Blue Cross, stressing always that the private practice of medicine is superior to any government controlled medicine. Create study groups among your auxiliary members and educate yourself. That is tremendously important! The American Medical Association is counting heavily on the medical auxiliary in the fields of public education. Our auxiliary is a liaison between the physician and the public. Let us develop a spirit of understanding and friendship between the laity and profession. We quote Dr. Edward Leroy Bortz, President of the American Medical Association, as saying, "The members of the Woman's Auxiliary excel in public relations, and the American Medical Association expects much from you this year in that respect. No group in the nation is so well qualified to speak for social aspects and personal contacts as are the women who know doctors better than anyone else in the world. The Bureau of Health Education is prepared to supply you with any type of material you may need. We recommend in particular the brochure on 'Voluntary health insurance versus compulsory health insurance.' We also recommend that you read 'The Road to Serfdom', published by the University of Chicago Press."

The Woman's Auxiliary to the Shreveport Medical Society on November 12, sponsored an open meeting for the public at the Louisiana State Exhibit Building. Participating in the round-table discussion of state voluntary prepayment medical care plan were Dr. H. Whitney Boggs of Shreveport, Dr. O. B. Owens of Alexandria and Mr. Frank Lais, Jr., of New Orleans.

Mr. Lais spoke on "The Doctor Assumes His Obligations" and explained the organization of the Louisiana Physicians Service and the prepayment surgical and obstetrical care plan. An insurance accountant, Mr. Lais was formerly associated with the Department of Insurance of Louisiana. He became affiliated with the State Medical Society over a year ago and assisted in the organization of the Louisiana Physicians Service. Also, he is executive director of the council on medical service and public relations of the Louisiana State Medical Society.

Every doctor's wife should consider it her personal responsibility to assist in promoting the prepayment medical care plan in her community. The success or failure of these plans may determine whether the medical profession will continue to control the practice of medicine or if it will be taken over by the government.

BOOK REVIEWS

Dermatologic Clues to Internal Disease: By Howard T. Behrman, M. D. New York, Grune & Stratten, 1947. Pp. 165. Illus. Price, $5.00.

According to the preface, the author's purpose in presenting this little book is to "correlate the manifestations of skin disorders with those arising in some visceral dysfunction."

Approximately 160 diseases are discussed alphabetically within 165 pages. Most of these conditions are disturbances of internal function with

either minor or major skin manifestations. Actually, the subject matter is too inclusive for such a small volume. Sample topics are: the avitaminoses, blastomycosis, coronary occlusion, ectopic pregnancy, endocrine disorders, erysipelas, gangosa, gonorrhea, hay fever, influenza, lupus erythematosis and yaws. With 118 black and white illustrations, most of which are good, taking space, there is little room left to do much more than list diseases. For example there are only six lines devoted to the subject of erythema multiforme. Much space is lost by listing the same disease under different names and stating that it is discussed under another heading.

While some readers will be disappointed, whether they be dermatologists or internists, the book does emphasize the fact that skin disturbances are often the reflection of internal disorder.

LEE DAVIDSON MCLEAN, M. D.

Surgical Pathology: By William Boyd, M. D., Dipl. Psychiat., Mr. R. C. P. Edin., F. C. C. P. London, LL. D. Sask., M. D. Oslo, F. R. S. C. 6th ed. Philadelphia, W. B. Saunders Co., 1947. Pp. 858. Illus. pl. Price, $10.00.

The content and form of the sixth edition of *Boyd's Surgical Pathology* are very much the same as the previous editions. The author presents the pathology in relation to symptomatology and the subject matter is accurate, concise and for the most part complete. The bibliography is excellent and the index adequate. Several additional subjects are discussed such as, tumors of the larynx, avitaminosis, fibrositis of the back, Bittner's milk factor, and the Papanicolaou stain. An interesting addition is the section on congenital heart disease. Everyone is familiar with the author's "easy-to-read" fluid style which is another factor in making this book outstanding. There is a definite improvement in the grade of paper resulting in much better illustrations of the excellent photographs and photomicrographs.

ERNEST G. DEBAKEY, M. D.

Hospital Care in the United States: Prepared by the Commission on Hospital Care, New York, The Commonwealth Fund, 1947. Pp. 631. Charts. Price, $4.50.

This book is the report of the findings and opinions of the Commission on Hospital Care, an independent public service committee, established by the American Hospital Association and composed of twenty members representing the fields of medicine, dentistry, hospital administration, public health, nursing, education, industry, agriculture and labor. The need for intelligent planning for the future development of hospital care programs is the main theme of this study. In addition, the necessity for the integration of hospital care with other medical care and social service facilities of the community is clearly indicated.

The report, which deals primarily with general hospitals, presents interesting data concerning the development and growth of hospitals as an institution for medical care. Relationships of hospitals to community life and organizations are also well analyzed. Information will be found in this book which is especially useful to (1) state hospital study groups which have been organized by state governments as the result of the Hospital and Survey Construction Act, Public Law 725, (2) hospital administrators, and (3) others who are interested in the general medical care program of the community. The many functions and services of the general hospital are discussed in detail, and pertinent problems of hospital management, such as size and distribution of hospitals, estimation of bed needs, training of professional personnel, and financing of hospital care, receive adequate attention.

This book is unique in that it sets forth, at the very beginning, a concisely stated summary of the conclusions and recommendations which resulted from the two-year study of the Commission on Hospital Care. Subsequent chapters give full discussions of the factors which led to these opinions reached by the Commission.

KIRK T. MOSLEY, M. D.

Stereoscopic Atlas of Neuroanatomy: By H. S. Rubinstein, M. D., Ph. D. and C. L. Davis, M. D. New York, Grune & Stratton, Inc., 1947. Pp. 19. Illus. pl. 43. Price, $7.50.

This atlas comprises 43 plates printed individually on cardboard (6⅞ x 9¼ inches) for convenience of viewing with the stereoscope. Each plate carries a pair of stereoscopic photographs printed in the bottom section, the area above being occupied by a fully labeled line drawing of the specimen. A 19-page booklet accompanies the atlas. It includes a guide for making dissections comparable to those illustrated and a subject index to the structures shown in the plates.

The first five plates in the series are devoted to the embryological history of the brain. The remaining plates illustrate surface features of the brain and gross structures exposed by dissection.

It was an excellent idea that led to the preparation of this atlas, for such a collection of photographs could serve a highly useful purpose. The execution, however, falls short of fulfilling the idea. Too many of the photographs are technically faulty, and in some instances the dissec-

tions themselves do not have the quality to be expected in a work of this kind.

HAROLD CUMMINS, Ph. D.

A Manual of Fractures and Dislocations: By Barbara Bartlett Stimson, A. B., M. D., Med. Sc. D., F. A. C. S. Philadelphia, Lee & Febiger, 1947. 2d., Pp. 223. Illus. Price, $3.25.

In the second edition of "A Manual of Fractures and Dislocations", Dr. Stimson has extensively revised the material presented. This revision is especially noticeable in the discussions of fractures of the humerus and fractures about the elbow. The text is written in a precise, almost cryptic, manner without exhaustive details, but with proper emphasis on both diagnosis and principles of treatment, which enhances its value as a guide for medical students. As a synopsis of fracture treatment, it fulfills its purpose admirably. If any criticism could be made, it would be directed toward the discussion of the time of immobilization of various fractures, which is included in the discussion under each separate fracture. The reviewer feels that there is a distinct probability of the students' getting the impression that a certain type of fracture is treated for such and such a period, and no more or no less. This criticism is extremely minor, considering the overall, thorough discussion of fundamentals of the diagnosis of fractures, bone repair in fractures, and the symptoms and signs of fractures, plus the thorough, if brief, discussion of the principles of treating fractures in general. This valuable guide for medical students should aid them in securing a pathway through the wealth of material published in text books or other larger texts on fractures.

JACK WICKSTROM, M. D.

Renal Hypertension: By Eduardo Braun-Menendez, Juan Carlos Fasciolo, Luis F. Leloir, Juan M. Munoz and Alberto C. Taquini. Translated by Lewis Dexter, M. D. Charles C. Thomas, 1946. Pp. 451. Illus. Price $6.75.

The contributions of the Argentine physiologists under the guidance of Houssay, to the present knowledge of experimental renal hypertension are set forth in this volume. The subject is developed in chronologic order, beginning with studies of the production of the hypertension of renal ischemia and terminating with a description of the homeostatic influence on blood pressure of the secretions liberated. Complete description of the nature and behavior of renin, hypersensinogen (renin activator) and hypertensin (angiotonin) is given including methods of their preparation.

The book contains a preponderance of the authors' experiments and deals other investigators short in some instances. It obviously will be of more interest to investigators than clinicians. Its large bibliography is helpful to those interested in the subject.

STANLEY COHEN, M. D.

Care of the Breast: By Else K. LaRoe, M. D. New York, Froben, 1947. Pp. 240. Illus. Price, $3.75.

This book is an endeavor by the author, Else K. LaRoe, M. D., to present the subject of the "Care of the Breast" in a lucid and comprehensive manner. The basic consideration is emphasis on certain antecedent pathologic manifestations which so frequently occur in cancer of the mammary gland and other chronic if not fatal, diseases. The embryology, anatomy, anomalies, tumors and reconstructive surgery of the female breast are all covered in this book which contains a total of 76 illustrations and 240 pages. A number of the illustrations are historic in character and a great part of the book is devoted to plastic surgery. Little space is given to radical surgical technic in cancer of the breast.

C. W. JOHNSON, M. D.

Synopsis of Allergy: By H. L. Alexander, A. B., M. D. New York, C. V. Mosby Co., 1947. Pp. 255. Price, $3.50.

The second edition of this compact summary clarifies nicely current ideas in the field of allergy. Much remains to be clarified however in our understanding of the complicated immunologic and physiologic processes that underlie allergy.

The most significant advances since the first edition of this book include the introduction of the antihistaminic drugs, work in vascular allergy, and newer conceptions of the relation of allergy to infectious diseases and of sensitization to drugs and chemicals.

To those desiring a synoptic view of allergy this book can be unreservedly recommended.

V. J. DERBES, M. D.

PUBLICATIONS RECEIVED

The Blakiston Company, Philadelphia: History of Medicine by Cecilia C. Mettler, A. B., Ed. B., A. M., Ph. D.

The C. V. Mosby Company, St. Louis: The Oculoratory Muscles, by Richard G. Scobee, B. A., M. D.

W. B. Saunders Company, Philadelphia: Gifford's Textbook of Ophthalmology (4th edition), by Francis H. Adler, M. D.; Pharmacology Therapeutics and Prescription Writing (5th edition), by Walter Arthur Bastedo, Ph. G., Ph. M. (Hon.), Sc. D. (Hon.), F. A. C. P.; A Textbook of Clinical Neurology (6th edition), by Israel S. Wechsler, M. D.

The Williams and Wilkins Company, Baltimore: An Atlas of Anatomy (2d edition), by J. C. Boileau Grant, M. C., M. D., Ch. B., F. R. C. S. (Edin.).